VOLUME 35 NUMBER 9 SEPTEMBER, 1952

AMERICAN JOURNAL
OF
OPHTHALMOLOGY

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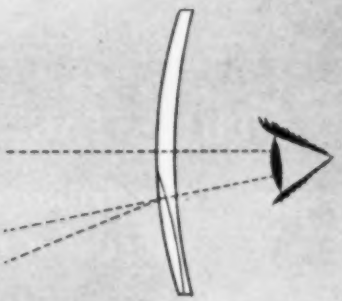

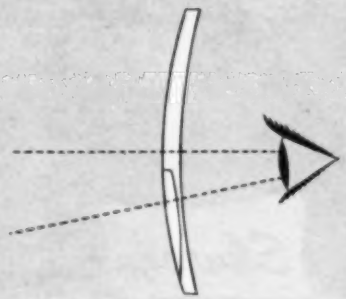

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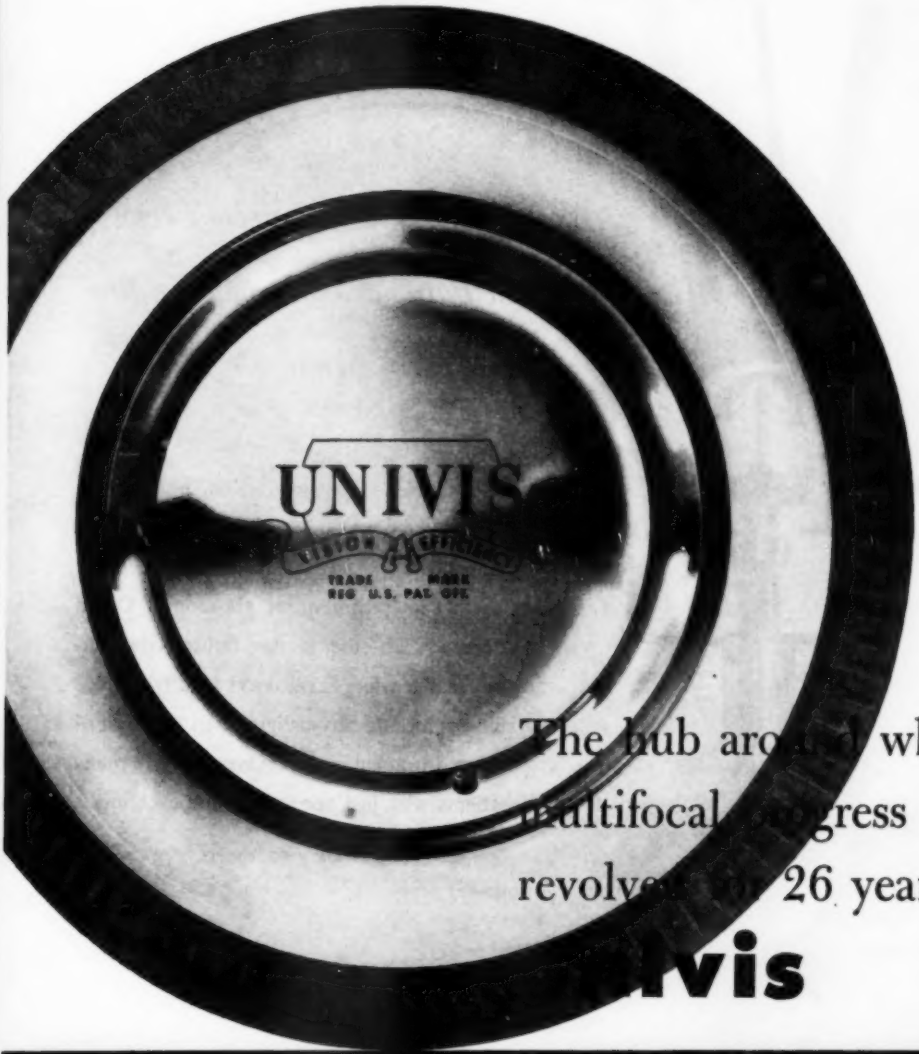

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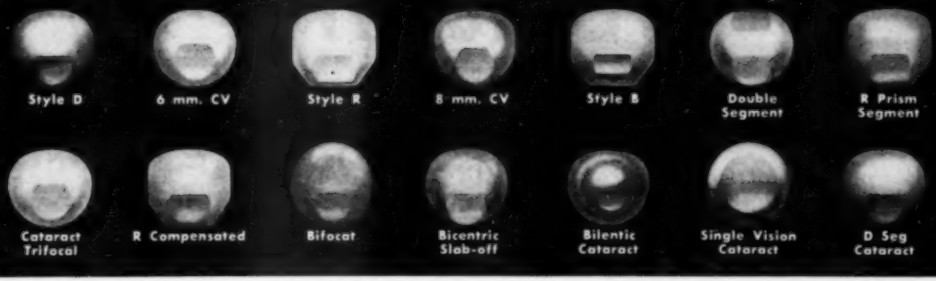

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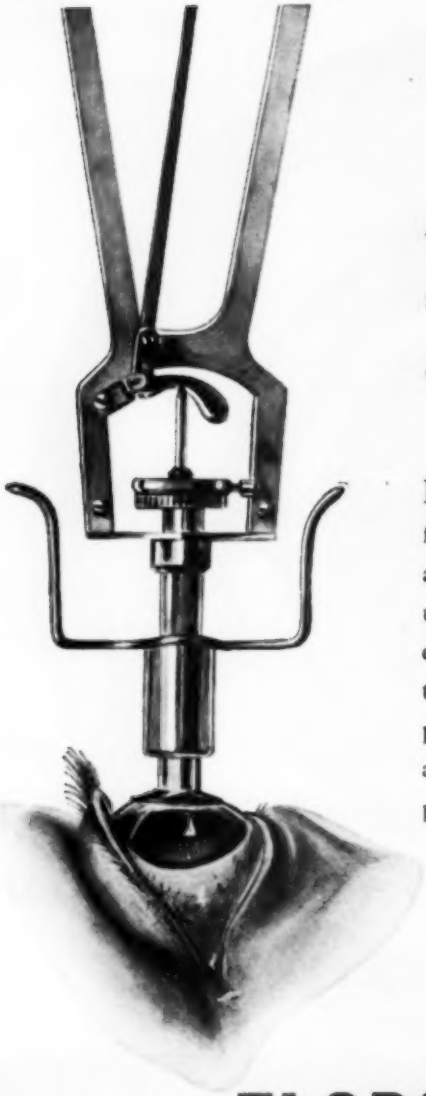

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1. *Mitsui, Y., et al.: Antibiotics and Chemotherapy 1:253 (July) 1951.*
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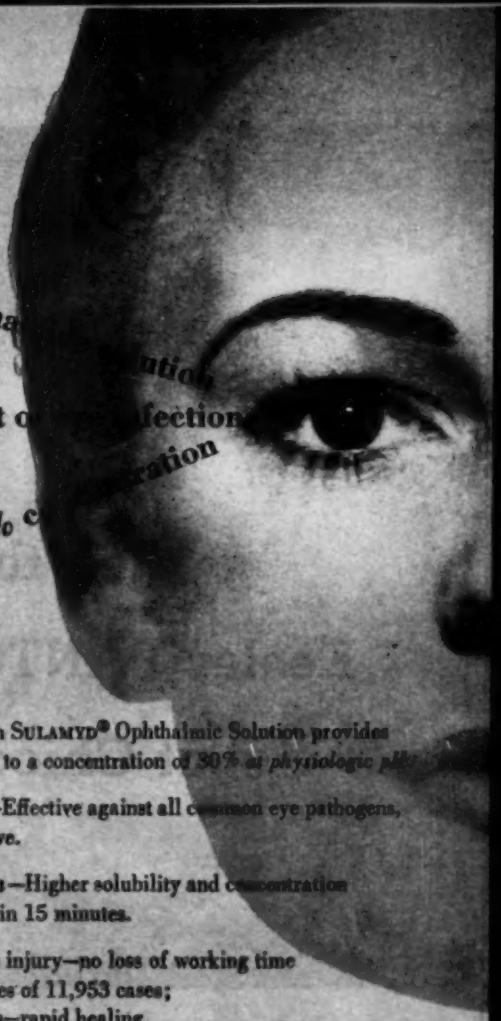

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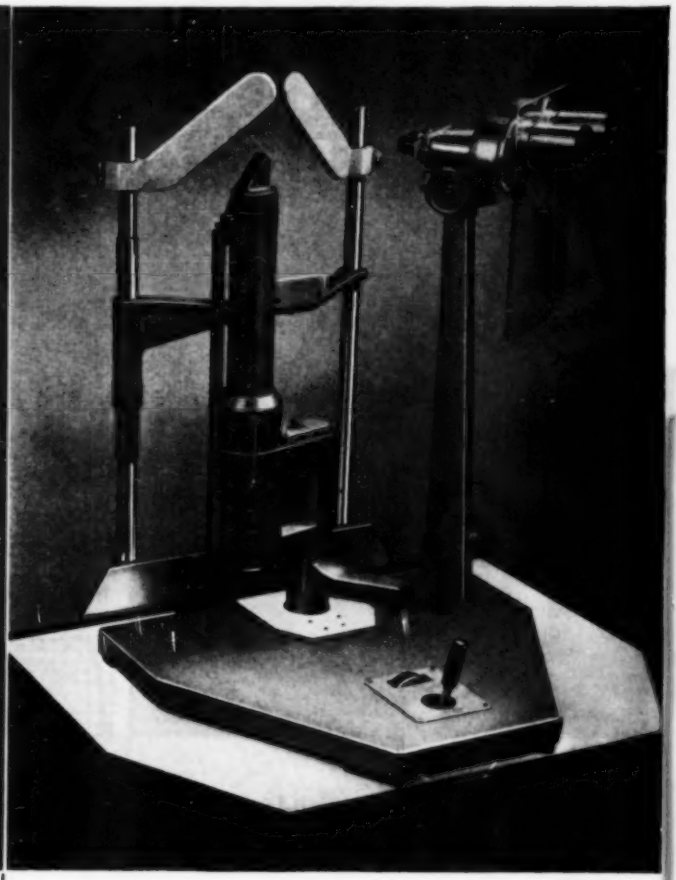

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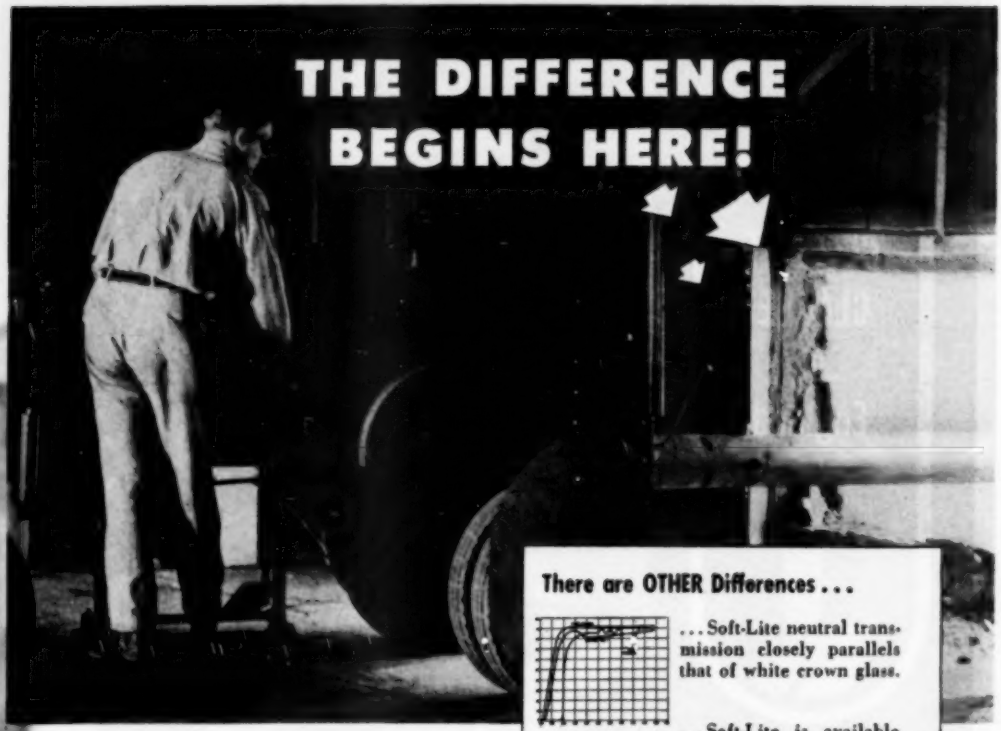

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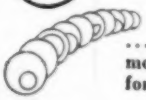

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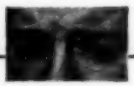
Some eyes can take it . . .  **some eyes cannot!**

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1. Scheie, H. G., Tyner, G. S., Buesseler, J. A., and Alfano, J. E., *J. A. M. A. Arch. Ophth.* 45:301, March 1951.
2. Leopold, I. H., Purnell, J. E., Cannon, E. J., Steinmetz, C. G., and McDonald, P. R., *Am. J. Ophth.* 34:361, March 1951.

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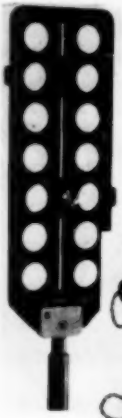

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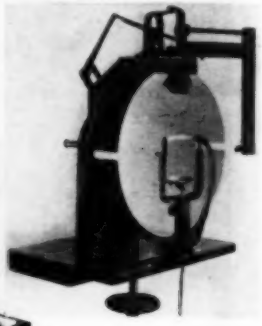

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ABSTRACTS

Diagnosis and therapy; Ocular motility; Conjunctiva, cornea, sclera; Uvea, sympathetic disease, aqueous; Glaucoma and ocular tension; Crystalline lens; Retina and vitreous; Optic nerve and chiasm; Neuro-ophthalmology; Eyeball, orbit, sinuses; Eyelids, lacrimal apparatus; Tumors; Injuries; Systemic disease and parasites; Congenital deformities, heredity; Hygiene, sociology, education, and history 1374

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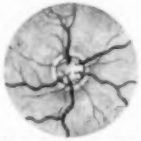

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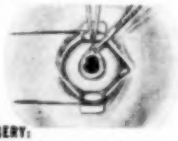

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VOLUME 35 SEPTEMBER, 1952 NUMBER 9

OCULAR CYSTINOSIS*

HARRIET G. GUILD, M.D., FRANK B. WALSH, M.D., AND RICHARD E. HOOVER, M.D.
Baltimore, Maryland

It is the purpose of this paper to present two cases of systemic cystinosis and to draw attention to the ophthalmic findings. So far as we are aware the eye findings of cystinosis have not been described in this country. However, they have been described elsewhere by Burki[1] in Germany and by Douglas[2] in England. The literature on the general subject of cystinosis has recently been covered by King and Lochridge.[3]

Cystinosis is a rare disorder characterized by the universal deposition of cystine crystals throughout cells of the reticulo-endothelial system. Most cases of cystinosis are characterized by chronic acidosis and renal glycosuria as may be seen in the type of "resistant rickets" described by Fanconi.

Since an intractable rickets is often the presenting complaint, this must be distinguished from (a) a simple vitamin D-resistant rickets in which rickets progresses in spite of adequate vitamin-D therapy, as a result of some failure of end-organ response to normal levels of vitamin D in the blood serum, and from (b) the ordinary renal rickets that occurs as a late complication of renal insufficiency in which the impairment of kidney function is a consequence of primary renal disease.

In the former there is no abnormality apart from the resistance to vitamin D and the rickets can be healed by the use of enormous doses. Furthermore, healing is maintained by continued therapy and no complications occur other than those related to the rickets itself.

In the latter, the rickets is a consequence of the phosphate retention and acidosis which develop in the course of progresssive renal disease when the kidney is no longer able to excrete phosphorus or to maintain an acid-base balance. This is a more complicated type of rickets in which there are often associated lesions of osteitis fibrosa cystica resulting from hyperparathyroidism, which occurs as the parathyroid gland attempts to compensate for the hypocalcemia induced by the high level of phosphorus in the blood serum. The resistance of this type of rickets to therapy is a natural consequence of the profound metabolic abnormality resulting from the basic renal disease.

The Fanconi syndrome (chronic acidosis, renal glycosuria, and hypophosphatemic rickets) first came to our attention 15 years ago (Guild, Pierce, and Lilienthal[4]). Since then, many cases have been reported and much has been learned regarding the disturbance. There is now a general agreement that the acidosis is an organic-acid acidosis resulting from an inborn error of protein metabolism.

Interest has become focused on the relationship between the Fanconi syndrome and cystine storage disease. Opinions differ as to whether the conditions represent different stages of the same disorder or whether they are merely related disturbances with a common denominator in the form of a congenital defect in amino-acid metabolism.

* From the Harriet Lane Home and the Wilmer Ophthalmological Institute of The Johns Hopkins Hospital and University.

Case reports

Case 1.

D. B., a white boy, was seen for the first time in January, 1948, at the age of 19 months, when he was admitted to the Harriet Lane Home for study because of intractable rickets. Family history was noncontributory. There was one normal sibling, aged 12 years, and the mother had had one miscarriage 11 years ago.

This child, born June 9, 1946, was delivered at term, weighing 7 lb. 13 oz., and was apparently normal until six months of age, gaining well and showing no unusual thirst or polyuria. Gradually thereafter excessive thirst was noted, urinary output increased, and the appetite for solid foods declined.

At nine months of age, swelling of the wrists was observed and a diagnosis of rickets was made. Oleum percomorphum, 10 drops daily, and vipenta, 20 drops, were substituted for the drisdol which had been given in a dosage of 10 drops daily since the age of three months.

By one year of age, the appetite had declined still further, vomiting became quite frequent and weight gain ceased. From time to time sugar was found in the urine.

Examination. When admitted to the hospital, the patient appeared as a very blonde, alert, intelligent little boy of 19 months, who was undersized and undernourished, measuring only 28 inches in height and weighing only 8.6 kg. He walked around the bed, calling for water, but speaking no other words that were intelligible.

The head was well formed with questionable slight bossing in the right parietal area and the fontanels were closed. Only eight teeth were present but these were in good condition. The eyes, including the fundi, were normal; hearing was not impaired; and the nasopharynx was normal except for signs of upper respiratory infection. Heart and lungs were clear, but blood pressure readings, not wholly satisfactory, were slightly elevated (112/85 mm. Hg).

Abdominal examination was negative. The extremities showed signs of rickets in the thickened epiphyses and slight bowing of the tibiae. There was also enlargement of the costochondral junctions.

Laboratory studies showed the following: Blood picture—normal. Urinalysis: Low specific gravity of 1.002 to 1.012 with pH of 7.5, trace to one-plus albumen, trace to one-plus sugar, no acetone, occasional pus cell and granular cast. Sugar in urine identified as glucose. Tuberculin test, 0.1 mg. and serologic test for syphilis, negative.

Blood chemistry determination: Nonprotein nitrogen 44 mg. percent, calcium 11.8 mg. percent, phosphorus 2.4 mg. percent, phosphatase 19.2 Bodansky units, total protein 7.8 gm. percent, CO_2 combining power 19.1 to 23.5 milliequivalents per liter, chlorides 100.4 to 115.6 milliequivalents, and sodium 137.5.

Glucose tolerance test: Fasting sugar 80 mg. percent; one-half hour, 160 mg. percent; one hour, 216 mg. percent; two hours, 112 mg. percent; three hours, 104 mg. percent; and four hours, 52 mg. percent. Phthalein excretion test: 15 minutes, 5.0 percent; 30 minutes, 10 percent; one hour, 15 percent; one and one-half hours, 10 percent; two hours, 10 percent; making a total of 50 percent in two hours. Urine culture—sterile.

X rays: Bones—changes characteristic of rickets. Intravenous pyelograms: bilateral excretion of the dye; pelves imperfectly visualized, but no gross abnormality. Concentration test (fluids withheld for 12 hours) resulted in acute dehydration with maximal specific gravity of 1.012.

Course. On the basis of the low-phosphorus rickets with relatively good kidney function, but with polyuria, polydipsia, borderline acidosis, and mild glycosuria, it was clear that the rickets was of metabolic origin. The Fanconi syndrome was suspected, although findings were not quite typical. Slitlamp examination of the eyes, requested with cystinosis in mind as an alternative possibility, was negative on January 17, 1948, and was not repeated on this admission. Studies of

the urine revealed large quantities of amino acids although the pattern, by paper chromatography, was not significantly abnormal and almost no cystine was present.

The child remained in the hospital for seven months while metabolic studies were in progress and while efforts were made to heal the rickets. During this period the blood nonprotein nitrogen was usually within normal limits, from 30 to 40 mg. percent with an occasional rise to 44 mg. percent and one reading of 50 mg. percent; the CO_2 combining power remained slightly low, averaging 19 to 22 meq./l.; the chlorides ranged from 96.2 to 115 meq. with most readings approximating 100, and the serum proteins were always normal.

Until treatment with vitamin D was started on April 25, 1948, the blood calcium remained at approximately 11 mg. percent and the phosphorus rarely exceeded 3.2 mg. percent, while the alkaline phosphatase approximated 30 B.U.

On April 23, 1948, vitamin D was started in a dosage of 60,000 units daily. By May 10, 1948, the phosphorus had risen to 5.4 mg. percent, and by May 19, 1948, there was evidence of healing in the X-ray films. On May 22nd, the dosage was reduced to 40,000 units daily and on June 22nd, when the calcium had risen to 15.7 mg. percent it was discontinued altogether.

The phosphorus level at that point was 4.4 mg. percent and the phosphatase was 12.8 B.U. By July 6, 1948, there was X-ray evidence of complete healing of the rickets. On this date the calcium was 12.1 mg. percent, phosphorus 4.0 mg. percent, and phosphatase 9.2 B.U. By August 4th the calcium had dropped to 11 mg. percent and the phosphorus to 3.0 mg. percent with a phosphatase of 13.8 B.U., although healing was still complete.

In spite of the healing of the rickets, other symptoms remained unchanged, and at the time of discharge on August 13th, the child weighed less than on admission (8.1 kg.) although he had grown two inches in length. Instructions were given regarding diet and fluid intake and vitamin D was to be resumed in a dosage of 20,000 units daily, alternating one week on and one week off therapy, with weekly determinations of serum calcium and phosphorus as a guide to further therapy.

The patient was not seen again for a period of almost two years. He then returned for reëvaluation with the story that vitamin D was continued as prescribed until December, 1949, when the special irradiated ergosterol with which he had been supplied at the time of discharge was exhausted. Mental development had proceeded normally during the interval, but the feeding difficulties, thirst, polyuria, and occasional vomiting had continued.

On readmission on April 11, 1950, the child, now almost four years of age, measured only 34 inches in height and weighed only 11 kg. but he was well proportioned. Although he was quite knock-kneed, there were no signs of rickets and he was obviously of normal intelligence. There was questionable slight haziness of the corneas but the eyes were otherwise normal externally and dentition was normal. The heart was normal with blood pressure, 75/55 mm. Hg; there was no enlargement of liver or spleen, and the remainder of the examination was negative.

Laboratory studies now showed marked impairment of kidney function. Urinary specific gravity was fixed at 1.002 to 1.006, reaction was usually alkaline and albumen was consistently present in a trace to one-plus concentration. Glycosuria had disappeared and the urinary sediment was negative.

Blood chemistry determinations showed nonprotein nitrogen 66 to 90 mg. percent; CO_2 combining power 16.6 to 22.5 meq. per liter; chlorides 92 to 99 meq., sodium 129 and potassium 3.6 to 4.6 meq. per liter; calcium and phosphorus (in spite of no vitamin D for four months) were normal or slightly elevated (calcium 9.9 to 11.6 mg. percent, and phosphorus 4.0 to 6.2 mg. percent with

phosphatase 6.4 to 13 B.U.); serum proteins normal, cholesterol slightly elevated, 240 to 364 mg. percent.

Phthalein excretion test showed only 15 percent excretion in two hours. Urea clearance, 25 percent of normal. Urine culture was negative. Retrograde pyelograms demonstrated an anatomically normal urinary tract.

X ray of the bones: Some osteoporosis but rickets still healed. X ray of chest and skull, normal. There was a moderate anemia but blood picture otherwise was normal.

Because findings at the time of the first admission had not been wholly typical of the classical Fanconi syndrome, and because healing of the rickets had been maintained in the interval with the development of renal insufficiency, a search was made again for evidence of cystinosis.

Slitlamp examination of the eyes this time showed characteristic glistening particles throughout both corneas. Bone-marrow aspirated from the sternum for microscopic examination was gritty and contained crystals that were morphologically compatible with those produced by cystine.

It was, therefore, assumed that precipitation of cystine crystals in the substance of the kidney accounted for the progressive renal insufficiency, with retention of phosphorus, as a consequence of glomerular damage, contributing to the healing of the rickets. The child's course was thus characteristic of that observed in proven cases of cystinosis.

With the diagnosis thus finally established, the child was discharged on symtomatic treatment only, to receive ordinary doses of vitamin D in the form of oleum percomorphum 10 or 15 drops a day and large amounts of vitamin C, as ascorbic acid, 400 mg. a day.

In June, 1951, a year after discharge, the patient was admitted to the hospital for the third time. He had been eating moderately well with other symptoms as usual, had grown one and one-half inches in height (now 35.5 inches), and had gained 1.1 kg. (weight now 12.1 kg.). For several months, however, he had shown photophobia and had complained of not seeing well. Vitamins had been supplied as visyneral, 15 drops daily.

On physical examination, with measurements equal to those of a child of two and one-half years, this five-year-old boy appeared fairly well nourished and was mentally alert. Photophobia was present, but the eyes were externally normal except for the slight haziness of the corneas noticed during the previous admission. The tonsils were large but there was no adenopathy. Heart was normal with blood pressure 85/50 mm. Hg., and lungs were clear. There was no enlargement of liver or spleen. Except for a rather marked genu valgum, the extremities showed no signs of rickets and the remainder of the examination was negative.

Laboratory studies showed a normal blood picture. Urinary specific gravity was fixed at 1.000 to 1.008, reaction was acid, albumen consistently one plus, and sugar consistently absent. Sediment also was negative.

Blood chemistry determinations showed nonprotein-nitrogen 62 to 98 mg. percent, calcium 12.5 to 13.5 mg. percent, phosphorus 5.2 to 9.0 mg. percent, phosphatase 13 B.U., Co_2 combining power 11.5 to 14.9 meq. per liter, chloride 100 meq./l, sodium 134.5 and potassium 3 meq./l. Phthalein excretion test: 4.0 percent in 15 minutes; 3.0 percent in 30 minutes; 7.0 percent at one hour; 5.0 percent at two hours, totaling 19 percent, approximately the same as on the previous admission. Urea clearance had fallen to 17 percent.

Glucose tolerance test: fasting sugar 120 mg. percent; one-half hour 276 mg. percent; one hour 288 mg. percent, two hours, 208 mg. percent; three hours, 160 mg. percent; four hours, 96 mg. percent.

Examination of the eyes showed no measurable impairment of vision but the child was uncoöperative. The photophobia seemed to be only that characteristic of a very blonde individual. There was a light haze of the cornea of each eye, but no vitreous opacities were seen and the fundi were normal. On slitlamp examination, myriads of

shiny crystalline particles were seen, as before, within the superficial stroma.

Examination of the bone marrow also again revealed numerous cystine crystals, but, as before, no cystine could be demonstrated in the urine. After vaccination against smallpox, the child was discharged without change in therapy, to be followed by his physican at home.

CASE 2

A. S., born November 19, 1948, a girl, was brought to the hospital on May 28, 1951, with complaint of renal rickets. Family history was noncontributory; a seven-month-old sister was normal. The patient was the result of the first pregnancy, which was uneventful. She was delivered spontaneously, weighing 8 lb. 3 oz.

Breast feeding was continued until five months; the diet thereafter was adequately supervised and she ate quite well until the age of 15 months. Vitamins were supplied as orange juice and 10 drops of oleum percomorphum daily until two years of age when polyvisol in a dosage of 0.6 cc. was added.

Early development was normal in that she sat alone at the age of six and one-half months, and spoke words at one year, followed by sentences at 20 months. Dentition was slightly delayed, with the first tooth at eight and one-half months, and she did not walk until 18 months of age.

This child's deviation from the normal was first noted at about one year when polydipsia became apparent. In retrospect, however, it was recalled that she had taken water and fruit juices more readily and in larger amounts than most babies do, since the age of five or six months.

At 12 to 15 months, it was also noted that she was voiding unusually large amounts, as the bed was very wet at night. At 15 months, her appetite declined, thirst became excessive (she would drink as much as 24 oz. of water before settling down for the night) and vomiting occurred quite frequently. From then on her weight remained stationary.

At about 18 months her appetite improved again, but polydipsia continued. At 21 months, an uncle, who was a physician, noted that the child was poorly nourished and undersized and referred her to a pediatrician in Nashville, Tennessee, who admitted her to the hospital there for study.

Physical examination at that time, at the age of two years, showed a child who was stunted in growth, measuring and weighing less than average for her age. The fontanel was still open, dentition was delayed, and the abdomen was markedly protruberant. X rays showed rachitic changes in the bones. The urine was of low specific gravity, the blood nonprotein nitrogen was 70 mg. percent and the polyuria was confirmed. Examination of the urine for cystine was negative. On the basis of the above findings a diagnosis of renal rickets was made, and the child was referred here for further study.

Examination on admission revealed an underdeveloped, fairly well-nourished little girl, age two and one-half years, measuring 31.25 inches in height and weighing 23 lb. 5 oz. (10.7 kg.). She was very alert and coöperative.

Skin was clear with a tinge of carotinemia and the hair was very fair. The forehead was prominent but there was no definite bossing. Eyes were externally normal and dentition was normal. There was an upper respiratory infection but no significant glandular enlargement. The heart was normal with blood pressure of 110/60 mm. Hg, and lungs were clear.

The tips of a normal liver and spleen were palpable; no other masses were felt. There was slight beading of the ribs, a marked knock-knee deformity, and slight thickening of the epiphyses. There was also a marked lordosis when the child stood. The remainder of the examination was negative.

Laboratory studies showed a normal blood picture, negative tuberculin test, and negative serologic test for syphilis. Urine was of low specific gravity (1.002 to 1.005), gave an acid reaction, and contained a trace to one plus albumen. A trace of sugar was present

in only one specimen; sediment, negative. Culture of the urine was sterile.

Blood chemistry determinations showed: Nonprotein-nitrogen 73 mg. percent, cholesterol 266 mg. percent, calcium 10.8 mg. percent, phosphorus 6.2 mg. percent, phosphatase 18.2 B. U., Co_2 combining power 11.5 meq./l, potassium 3.6 meq./l, total protein 8.4 gm. percent, albumen 5.9 gm. percent, globulin 2.5 gm. percent.

Phthalein excretion test: 15 minutes, 2.5 percent; one hour 8.0 percent; two hours, 5.0 percent; two-hour excretion, 15.5 percent.

Glucose tolerance test: Fasting sugar, 120 mg. percent; one-half hour, 200 mg. percent; two hours, 152 mg. percent; three hours, 136 mg. percent; 4.5 hours, 152 mg. percent. There was a trace of sugar in all but the fasting specimen of urine.

X-ray of the chest was negative except for evidence of slight flaring of the ribs; the long bones showed healing rickets of moderate degree.

This little girl's history, together with the evidence of a refractory rickets now healing, was so suggestive of cystinosis that she was referred for slitlamp examination and a bone-marrow aspiration was done before any further investigation was planned.

The slitlamp examination showed the characteristic picture demonstrated by the other child, and bone-marrow examination also confirmed the diagnosis. The importance of these examinations in cases in which cystinosis is suspected is emphasized by the failure to demonstrate cystine in the urine of these patients, a paradox that is quite usual.

Comment

Our cases have been described sufficiently fully to outline the entire symptomatology usually associated with or characterized by cystinosis. We may now proceed to a consideration of the ophthalmic features.

External examination revealed only slight haziness of the corneas without evidence of inflammation or vascularization. In our wo cases there was a single important clinical finding which could be determined only by slitlamp examination. Myriads of refractile rod-or needle-shaped particles were seen in both corneas. These particles were uniformly distributed throughout the corneas of both eyes. They were situated in the superficial stroma and were possibly more numerous toward the center of the cornea.

Certainly no crystals were observed in or near Descemet's membrane. Whether or not Bowman's membrane contained crystals was hard to determine because, in these children whose ability to coöperate was limited, examinations with the slitlamp were difficult.

Crystals were not identified in the conjunctiva but well may have been present and overlooked. So far as we could determine the irises, lenses, and vitreous were normal and contained no crystals.

Ophthalmoscopic examinations showed the fundi to be normal. It is noteworthy that we were unable to detect the crystals with the ophthalmoscope or with a handlamp.

Burki[1] described a case which at autopsy showed the characteristic widespread deposition of cystine crystals in many organs. Crystals were found in the conjunctiva, corneas, uveal tract, episclera, and in small amounts in the sclera, but none were seen in the lens, vitreous, and retina.

Crystals were present in the kidney, spleen, lymph nodes, liver, supporting tissue of the lung, gastrointestinal tract, thymus, testes, and bone marrow. They were absent in the thyroid, pituitary, pineal, brain, and cord. A few were found in the pia mater.

Douglas[2] in reporting the case of a girl, aged six years, saw corneal crystals as described in our cases. Also, he observed crystals in the palpebral and bulbar conjunctiva and in the fornices. The irises were lightly powdered with crystals. He remarked upon five such cases of cystinosis having been observed in a children's hospital clinic.

It would not add to the value of this paper to attempt any classification of corneal crystals either on the basis of refractility, size and shape, or chemical composition. Our

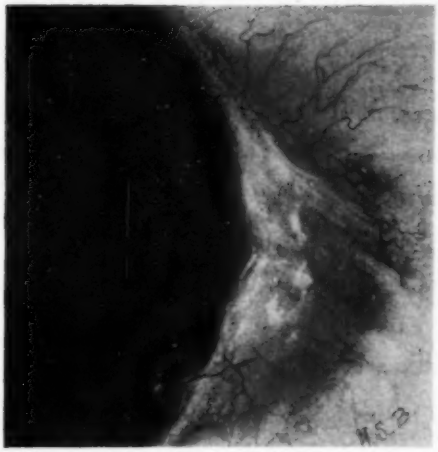

Fig. 1 (Guild, Walsh, and Hoover). Hypercalcemia. (From Walsh, F. B., and Howard, J. E.: J. Clin. Endocrinol. 7:644-652 (Sept.) 1947.)

cases described here probably fit properly into Fry's[5] third group—namely, crystalline degeneration secondary to local or generalized disease. Fry's[5] other categories are hereditary and nonhereditary crystalline dystrophy.

calcemia is mentioned here because cases characterized by cystinosis require intensive vitamin-D therapy which may result in very high blood calcium (Case 1). Conceivably this could result in calcium deposits in the cornea and conjunctiva. The slitlamp picture of hypercalcemia is entirely different from that of cystinosis. Compare Figures 1 and 2.

Other corneal affections might be confused with cystinosis if slitlamp examination is omitted—for example, Hurler's syndrome, band-shaped keratitis (hypercalcemia one cause), corneal changes seen in Still's disease, and seemingly cases associated with high blood cholesterol as described by Toth.[8]

Conclusions

1. On the basis of two cases herein described it has been shown that the diagnosis of cystinosis can be established by slitlamp examination of the corneas and microscopic examination of the bone marrow.

2. Examination of the bone marrow might provide interesting information in adults

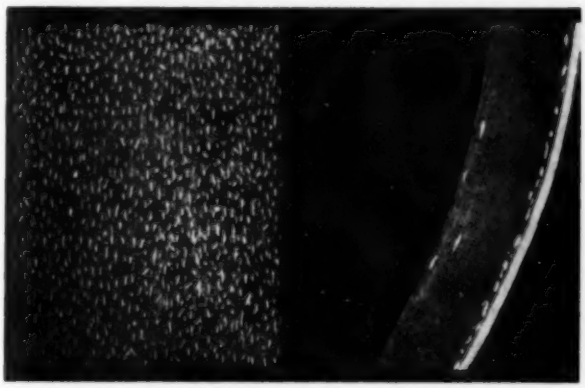

Fig. 2 (Guild, Walsh, and Hoover). Cystinosis of the cornea.

Within recent years it has been shown (Walsh and Howard,[6] Cogan[7]) that hypercalcemia in some instances has a specific eye picture which is best identified by use of the slitlamp. This ocular manifestation of hyperexhibiting so-called crystalline dystrophy of the cornea. Such cases might be incomplete forms of cystinosis.

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STUDIES ON THE EFFECT OF HIGH OXYGEN ADMINISTRATION IN RETROLENTAL FIBROPLASIA*

I. Nursery observations

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In the Gallinger Municipal Hospital from January, 1948, to January, 1951, 18 of 21 infants who developed retrolental fibroplasia received prolonged oxygen therapy at high concentrations. High oxygen therapy could not be accepted as a causal factor, however, since the smaller infants generally received more oxygen. To evaluate the possible role of oxygen therapy in retrolental fibroplasia, a controlled oxygen-administration program was instituted. An analysis of the first of this three-year study is presented.

Subjects and Methods

All infants included in this investigation were delivered in the Gallinger Municipal Hospital obstetrical division. Infants with birth weights under 3.5 lb. were placed in one of two groups on admission to the premature nursery. Group I was maintained in high oxygen (65 to 70 percent) for four to seven weeks. Group II received lower oxygen (under 40-percent concentrations). The nursery routine was otherwise identical in the two groups.

Infants were placed in high or lower oxygen levels on an alternate admission basis regardless of birth weights. Oxygen tensions were controlled by incubator samplings at eight-hour intervals with the electronic analyzer (accurate within two-percent concentrations).

In general, approximately, seven liters for the high-oxygen levels and approximately two liters for the lower levels gave the desired concentrations in the isolette-type incubators. However, when the incubator concentration was found below 60 percent, the flow rate was raised, or when above 35 to 40 percent in the lower oxygen group, the flow rate was reduced.

The infants in high oxygen were maintained at these levels constantly for from four to seven weeks. Weaning off from high oxygen was done by gradually reducing the incubator concentrations over a period of one week. The infants in lower oxygen tensions were kept in incubators for from 24 hours up to two weeks at levels under 40 percent. Weaning off from lower oxygen was done over one to three days. The infants in each group were examined ophthalmoscopically

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TABLE 1

RESULTS OF STUDY OF 65 CASES

Oxygen Levels	Total Number of Infants	Normal Eyegrounds	Retrolental Fibroplasia			
			Grade			
			I	II	III	IV
High	28	11	3	7	2	5
Low	37	31	4	2	0	0

at regular intervals and followed after discharge in a special clinic until they were six months of age.

A brief resume of pertinent nursery routine shows that 5.0 mg. of vitamin K were administered routinely on the first day. The smaller infants usually received nothing by mouth for the first 36 to 48 hours, at which time they were started on sterile water. The larger infants were started at 12 to 24 hours.

On about the third day a half-skimmed milk formula with cartose syrup was begun and increased gradually. When the infant weighed about 4.5 lb., a straight evaporated-milk formula was started. Starting at two weeks of age, 20 drops of visynerol multivitamin preparation was given daily to all infants. This water-miscible preparation contains vitamins A, B complex, C, and D. Starting at 30 days, 60 mg. of ferrous sulfate was given twice daily.

Transfusions were administered during this study in only two cases, neither of which developed retrolental fibroplasia. One infant in the high-oxygen group who developed retrolental fibroplasia was given ACTH and is not included in the analysis of cases. No vitamin E was administered in this study.

ANALYSIS OF CASES

In the first year of this investigation, 76 infants with birth weights under 3.0 lb. 8.0 oz. were studied. Eight cases are deleted in this analysis because incubator oxygen levels were not sufficiently constant. None of these eight infants progressed beyond Grade-II changes. Three infants who were normal in the nursery did not return for follow-up examinations after discharge and are deleted. The results in the remaining 65 cases are tabulated in Table 1.

Owens and Owens'[1] classification of retrolental fibroplasia was slightly modified to include a fourth stage of completely arrested disease with residual retrolental membranes. Stage I represented marked dilatation and tortuosity of the retinal vessels with occasional areas of mild retinal edema. Stage II showed vascular changes, increased retinal edema, retinal hemorrhages, and vitreous clouding and opacities. Stage III represented detachment of the retina, new vessel formation, retinal hemorrhages, and vitreous opacities. Stage IV represented the arrested phase of the disease at total or almost complete permanent retinal membrane behind the lens.

Grade III and Grade IV retrolental fibroplasia occurred in seven cases (25 percent) receiving constant high oxygen. None of the 37 infants in the lower oxygen group progressed beyond Grade II changes. It is significant that infants who received prolonged high-oxygen levels were not necessarily the smaller infants. A continuous high or lower oxygen regime was administered on an alternate admission basis instead of the usual procedure of giving more oxygen to the smaller infants.

Early in our study, Gordon[2] pointed out to us that a similar investigation being carried out at the University of Colorado showed that premature infants thrive with much lower oxygen-environment concentrations than had been commonly advocated.

DISCUSSION

To evaluate oxygen administration criti-

cally it is essential that incubator oxygen concentration be measured frequently with an accurate oxygen analyzer. Flow rates as recorded in liters per minute do not necessarily indicate the incubator oxygen levels.[3]

Graham and associates,[4] in a carefully performed experiment on 44 premature infants, found a close correlation between incubator oxygen levels and arterial oxygen saturations. It is significant that at room oxygen (20 percent), they found arterial oxygen saturation to average 93 percent; at 30 to 55 percent, it averaged 96 percent; and at 70- to 79-percent oxygen concentrations, 100 percent arterial saturation was recorded.

Theoretically a gross pulmonary or circulatory anomaly could lower arterial oxygen values; however, careful clinical examinations should reveal these exceptional cases. In the remainder of our studies, hemoglobins, hematocrits, and simultaneous arterial oxygen saturations and incubator concentrations will be performed.

The toxic effects of hyperoxia in man and animals are well established and the monographs of Stadie and associates[5] and Bean[6] review the extensive studies that have been performed. Adult animals in 80-percent oxygen typically show oxygen poisoning with acute pulmonary edema developing in from three to eight days. Young rats, rabbits, and mice have been reported to be relatively immune to sustained high oxygen levels. It is significant, however, that in dogs maintained at 80-percent oxygen level, Paine[7] noted acute oxygen poisoning occurring in young puppies as readily as in adult dogs.

The following observations suggest mechanisms by which increased oxygen tensions might influence the developing premature retina.

1. In four cases of retrolental fibroplasia we have noted marked attenuation of the retinal vessels occurring prior to the development of dilatation and tortuosity. This narrowing has also been observed by Lamott[8] in approximately one third of his cases that developed retrolental fibroplasia. We have also noted in premature infants a visible narrowing of the retinal arteries and veins after one hour when increasing oxygen from 20 to 80 percent.

This is consistent with the report of Cusick and others[9] who reported an average of 25-percent constriction both in the retinal arterioles and retinal veins in adults after breathing pure oxygen.

Tinel,[10] Wolff and Lennox,[11] Cobb and Fremont-Smith,[12] and Schmidt,[13] describe a constriction of the pial or superficial cerebral vessels in animals breathing high oxygen. Bernthal[14] and Bronk and Gessell[15] found in animals that administration of 50-percent oxygen decreased the carotid blood flow. Ketty and Schmidt,[16] using the nitrous-oxide technique, demonstrated a reduction in cerebral blood flow in humans breathing 85-percent to 100-percent oxygen.

By diminishing retinal blood flow high oxygen might diminish the supply of other necessary metabolites to the premature retina.

2. There are considerable experimental data[17, 18] pointing to a destruction of intracellular enzymes, particularly dehydrogenase systems of the brain, in oxygen poisoning. Cusick[9] suggested that the retinal vessel constriction noted on breathing high oxygen is a mechanism to reduce retinal blood flow in an attempt to protect the retina from excessive oxygen levels. The mechanism is apparently inadequate as the color of the retinal veins approximates that of the arterioles when breathing high oxygen. Conceivably prolonged high oxygen administration might alter retinal enzyme systems in the premature.

3. Studies on the fetus in utero show that oxygen saturation of arterial blood is approximately 50 percent.[19] After birth, the arterial oxygen saturation rises in a few hours to approximately 90 percent in room atmosphere.[19] The final maturation of the premature infant, therefore, occurs at arterial oxygen saturations of 90 percent plus; whereas, in normal gestation, the fetus would have continued to develop in utero at considerably lower oxygen tensions.

Abnormal proliferation of the developing

retina might result from these relatively higher oxygen tensions. The transfer of incubator oxygen across the cornea to the premature retina is an undetermined factor that may further increase oxygen tensions in the retina.

It must be realized that there is no experimental proof to show that diminishing the retinal blood flow, altering the retinal enzyme systems, or exposing the developing retina to relatively higher oxygen tensions can produce the changes of retrolental fibroplasia. These mechanisms only suggest possible methods by which elevated oxygen tensions might influence the developing premature retina.

Szewczyk[20] removed prematures from incubators and recorded dilatation of retinal vessels, hemorrhages, and edema within the first few days out of oxygen. On replacing infants with early retrolental fibroplasia in oxygen he observed a uniform regression of the disease.

Our cases in continuous high oxygen seemed good subjects to test his observations. Two infants were studied.

Infant A weighed 2.0 lb., 7.0 oz. at birth and Infant B, 2.0 lb., 9.0 oz. Each had been in approximately 70-percent oxygen for 32 days.

Examination immediately prior to removal from the incubator revealed dilation and tortuosity of the retinal vessels, vitreous haze, and an index myopia of approximately six diopters in each infant. Infant B showed in addition localized areas of retinal edema along the vessels.

Instead of being gradually weaned off from oxygen, as is done routinely, these infants were suddenly removed from constant 70-percent oxygen to room oxygen (20-percent). Infant A required supplements of oxygen for one-half hour after meals for the first 48 hours due to slight respiratory distress. Infant B required supplementary oxygen on two occasions after feeding.

The eyes were examined daily for four days, then at two-day intervals to the 10th day, then once weekly for 60 days.

Infant A showed essentially no change in the eyegrounds in the first four days. In subsequent examinations, a regression of the retrolental fibroplasia with clearing of the vitreous and attentuation of the dilated vessels was observed.

Infant B showed a slight but definite increase in caliber and tortuosity of the arteries and veins during the first four days out of high oxygen. One solitary hemorrhage was noted in the nasal periphery of the left eye on the third day out of high oxygen. This hemorrhage cleared and the retinal vessels gradually had resumed normal size 21 days after removal from the incubator. The eyes have remained normal in both cases up to the age of three months.

These two infants demonstrate a spontaneous regression of mild retrolental fibroplasia to normal in spite of the anoxic insult that resulted from sudden withdrawal from a continuous 70-percent oxygen environment. These observations raise the question of how many of Szewczyk's[20] cases with early retrolental fibroplasia would have regressed to normal regardless of the oxygen environment.

Retrolental fibroplasia changes were detected in some of our cases while still in incubators with high oxygen. Also weaning off from high oxygen was done gradually over seven to eight days. These observations suggest that prolonged high oxygen, if injurious, results from a direct effect instead of from the relative anoxia that might result from the too rapid withdrawal from oxygen as cited by Szewczyk.[20]

If prolonged high oxygen therapy can be conclusively correlated with the incidence of retrolental fibroplasia, other factors may play a basic role in the disease's pathogenesis. Six cases of mild retrolental fibroplasia were recorded in our lower oxygen group. A theory based upon increased arterial oxygen tensions relative to those sustained in utero could, however, provide for occasional cases of retrolental fibroplasia developing when no supplementary oxygen is administered postnatally.

The data cited here together with the sur-

veys of Campbell,[21] Ryan,[22] Kinsey and Zacharias,[23] and Crosse,[24, 25] suggest strongly that high oxygen administration is a factor in the pathogenesis of retrolental fibroplasia. However, in view of the bizarre manner in which the incidence of the disease fluctuates, additional rigidly controlled observations are necessary to establish this concept. There are now sufficient data to question the advisibility of the "routine" use of prolonged high oxygen concentrations in the nursery.

Summary

1. The results of the first of a three-year controlled oxygen nursery study are cited. Seven of 28 infants receiving prolonged high oxygen levels progressed to Grades III and IV retrolental fibroplasia. Of 37 infants in the lower oxygen group none progressed beyond Grade II changes.

2. Mechanisms by which prolonged high oxygen administration might influence the premature retina are discussed.

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THE PHYSICIAN'S OPPORTUNITY IN PREVENTIVE OPHTHALMOLOGY*

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Clinical application frequently lags behind the accumulation of scientific data. It is therefore fitting, at varied intervals, to see how we can best utilize existing knowledge. The importance of this in the field of preventive ophthalmology can be realized when we learn that there were 22,000 new cases of blindness in 1950—2,000 in the state of New York. The opportunities afforded physicians in this field can best be grouped according to the various medical specialties.

INTERNISTS

Routine vision records should be included in every complete physical examination. If the vision is not normal with glasses, an ophthalmologic examination is indicated unless the patient is already under the care of an oculist.

Syphilis. Freeble and Donohue[1] in a study of the cause of blindness in Ohio found syphilis first in the 10 leading causes; 7.9 percent were due to this factor. Internists should do a Wassermann test routinely in every complete examination of a patient, remembering that in order to avoid late occurrences of neurosyphilis, intensive treatment in the first stages of luetic infection should be given.

Of the pathologic lesions in luetic blindness, optic atrophy is by far the greatest primary factor (67.7 percent).

J. V. Klauder and his co-workers[2] have rightly emphasized that with penicillin or induced malaria, contracted fields of optic-nerve atrophy in central-nervous system syphilis can disappear if given before the discs show pathologic paleness, the preatrophic or inflammatory stage of Bruetsch—in contrast to the atrophic stages in which the changes are irreversible and the goal is merely to retard the progress of the disease.

The problem is how can one make an early diagnosis?

The interval between infection and optic-nerve atrophy is rarely less than 10 years, the average being 18 years. One should realize that contraction of the peripheral fields precede the loss of vision and pallor of the optic disc. Repeated field studies at regular intervals should be done before eye symptoms appear on all patients with lues of the central nervous system.

Pupillary abnormalities are among the earliest and most constant signs of neurosyphilis—preceding by years the appearance of subjective symptoms. Their detection constitutes a short cut to the clinical diagnosis of neurosyphilis.

Pupillography may give positive signs of afferent pathway involvement before gross pupillary abnormalities can be discovered. Lowenstein reports that, if one nerve is involved, pupillography will show a lowered reaction in the involved side before field or fundus changes. This consists of a reduced quantity of light that comes to the third nerve nucleus on the affected side. Anisocoria

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may be absent. This test shows involvement of the afferent pathway but does not differentiate optic nerve from optic tract lesions nor does it differentiate etiologic factors.

Glaucoma prevention and detection. Systemic belladonna and its alkaloids have been known to induce glaucoma in susceptible individuals. One should inquire into the family history of glaucoma and question about visual disturbances before prescribing atropine systematically or instilling mydriatics for routine fundus study. In the latter case, the pupil should be contracted with pilocarpine before allowing the patient to leave the office.

For earlier detection of glaucoma, the use of the Berens-Tolman tonometer by the internist should be encouraged. The internist should also think of glaucoma when a patient has headache and vomiting and inquire about the vision; it may be that the condition is acute glaucoma rather than an acute cholecystitis.

Digitalis and quinine occasionally affect vision. Digitalis may produce xanthopsia and a retrobulbar neuritis with central scotoma. With quinine, amblyopia may occur accompanied by haziness of the disc margins, constriction of the retinal arteries, and pallor of the disc.

In prescribing reducing diets one should include essentials and not induce nutritional amblyopia.

By the same token, new drugs for weight reduction should be well investigated before released for general use. One has only to think back to 1941 when Horner[3] collected from the literature 177 cataracts produced by dinitrophenol. Many times that number probably were not reported. Brain has reported hyperthyroidism induced by the taking of thyroid to lose weight. An exophthalmos so produced not infrequently starts unilaterally.

Eye complications in diabetes are more in the province of the internist than the ophthalmologist. The extreme importance of careful treatment by diet and insulin is more completely understood when it is recognized that they are the only means we have of preventing or at least retarding retinal complications. It is the internist who should detect diabetics before symptoms appear by ruling out diabetes in every patient and, when diabetes is once diagnosed, to see the patients at regular intervals—warn them of the importance of close adherence to diet to postpone eye involvement—and lastly, but most important—keep them on the proper regime, namely a low calorie, well-controlled diet to prevent glycosuria and not the "free diet" advised by some.

Howard Root[4,5] of the Joslin Clinic, in a study of 400 cases of diabetes of 10 to 25 years' duration, found diabetic retinopathy in 82 percent of patients on a free diet compared to 44 percent of those on a controlled diet.

With proper dietary control, progression of retinal lesions may be retarded sufficiently to permit normal vision for 30 or more years. Reuting[6] reported on 50 diabetics followed 25 and more years—the average 28.4 years. No instance of blindness occurred in his series. These cases were all well controlled.

Sherrill[7] reported on 40 patients who had had diabetes 20 to 37 years—some were under his care for more than 30 years. None had visual disturbances, although there were minor retinal changes. There was no kidney involvement, and there were 10 successful pregnancies in seven of these individuals. They avoided obesity and followed a low calorie regime and prevented glycosuria. Twenty-eight patients studied by the membership of the New York Society for Clinical Ophthalmology had diabetes for 25 years and more—75 percent had 20/40 or better vision and three had 20/20, O.U.

When we realize that most of the 25-year patients were on high-fat diets during the early years of their disease and insulin was inadequate, it is safe to prophesy that much better end results will be obtained in the next 25 years than the past 25 years.

As to high-fat diet, the Newburgh-Marsh high-fat regime of 30 years ago received sufficient trial over a number of years to show

that early complications followed its use.

As to uncontrolled diabetes, we know that there is a disturbance of normal nitrogen balance which is responsible for the low plasma proteins found in diabetes of long standing. The relationship of low plasma protein to diabetic retinopathy has been sufficiently emphasized. Mosenthal also recalls the importance of conserving body protein which is destroyed in the presence of glycosuria. The effect of chronic dehydration associated with glycosuria is a factor in tissue injury. It is known that the degenerative changes in the beta cells of the islands of Langerhans associated with hyperglycemia are reversible when normal glycemia is restored.

Microaneurysms in the diabetic retina were described as far back as 1876 by MacKenzie.[8] More recently Ballantyne in 1943 and Friedenwald stressed that these lesions are the basis of many round red spots in the diabetic retina. They are not pathognomonic of diabetes however, as Ashton[9] has shown that, of 89 eyes removed at operation in nondiabetes, 33.7 percent revealed the presence of microaneurysms, the majority associated with glaucoma. Of 85 eyes examined at post mortem in nondiabetics, 34.1 percent showed aneurysms. In these latter diseases the pathologic process in the retina is not necessarily progressive. We can take heart, therefore, particularly when we know that the changes are reversible.

Sherrill reports a case of a woman, aged 26 years, with diabetes of 18 years' duration. "Myriads of round discrete aneurysms were noted in the perimacula area of both eyes during her pregnancy. The latter was terminated in the eighth month and within a month only a few 'red dots' could be seen."

O'Brien and Allen[10] showed that retinopathy disappeared after two months of good control in six diabetic individuals, all of whom were under 31 years of age. Capillary dilation in the conjunctival vessels especially in the plica semilunaris may be of diagnostic importance.[11] (See Figure 1.)

Fig. 1 (Givner). Capillary dilation in the plica semilunaris in a case of diabetes of 33 years' duration.

Diabetic retinopathy may be found in the absence of Kimmelstiel-Wilson disease but not vice versa. Both conditions are probably of the same etiologic relationship—poorly controlled diabetes. Both have capillary dilation and proliferation of the vessel walls. There are hyaline deposits in the intercapillary connective tissue of the kidney on the one hand and waxy exudates in the retina in the other; Allen believes the lesions arise within the capillary wall of the kidney and are not intercapillary as considered by Kimmelstiel. Friedenwald theorized that the dilated capillaries found in the kidney may be capillary microaneurysms of similar origin to those in the retina.

Anesthetists

Inadvertent pressure on the eye during closed mask anesthesia has induced central retinal artery occlusion in patients who also sustained blood loss and shock.[12] (See Figure 2.) The affected eye will be on the side on which the tubes lead from the tank to the mask. The tubes exert more pressure on that side. Rundle, in 1947, showed that exophthalmos of 2.25 mm. occurs during anesthesia. With the mask in place and with exertion

Fig. 2 (Givner). Sharp line of demarcation between the involved and uninvolved portion of the retina.

of pressure, a normal eye can easily have its intraocular pressure raised to 54 mm. Hg (Schiøtz). If one applies pressure to a normal eye of a rabbit for 20 minutes, the circulation can be stopped and edema of the retina and cornea and segmentation of the blood columns in both arteries and veins in the retina can occur. On release of the pressure, hemorrhages appear between the disc and the macula.

Given a low nasal bridge, the possibilities of such pressure are greater. This factor in a patient that sustains loss of blood and shock —conditions that contribute to sludging of the blood—may initiate the closure of the central retinal artery. That this accident is not a rare one is attested to by the fact that I now have knowledge of 12 cases in which it occurred.

Corneal abrasions are also commonly produced. Loss of epithelium may occur after ether is dropped into the eye.

All of these mishaps are preventable.

Surgeons

Great care and much consideration should be given to patients of menopausal age, who show eye signs but very few systemic signs of hyperthyroidism, before carrying out thyroidectomy either by surgical or medical means (propylthiouracil or radioactive iodine). Means called these cases "disassociation of thyrotoxicosis and ophthalmopathy." Malignant exophthalmos may too often be induced. Laurence Sloan states "any sudden diminution in thyroid function while the eye changes are unstable may precipitate severe progressive malignant exophthalmos."

Dermatologists

Stevens-Johnson disease. Loss of vision in Stevens-Johnson disease is frequently due to neglect of the eyes until corneal complications have already occurred. When the internists or dermatologists consult the eye man only after damage has been done, the corneal ulcer or conjunctival infection may be shown to be due to a secondary invader, namely the Staphylococcus aureus.

It is quite possible that the etiologic agent of erythema multiforme exudativa, if it is a virus, may sensitize the body for action by the staphylococcus or, if the causal agent be drugs or allergies, reduce the vitality of the tissues so that organism acts where usually it would not.

In all cases of this disease in which bacteriologic studies have been done on the conjunctiva, cornea, or vitreous in panophthalmitis, Staphylococcus aureus has been constantly found even though staphylococcus in general is present in only 64 percent of normal conjunctivas. By means of cleanliness and use of antibiotics and the like, these cases of pseudomembranous or purulent conjunctivitis, which in the past caused loss of vision and occasionally blindness, can many times be held in check.

Atopic dermatitis. A small percentage of cases have associated detachment of the retina. Karl Mylius has stressed vasomotor disturbances and abnormal reaction of the sympathetic nervous system to explain it. It is possible that some detachments are caused by the patient. The dermatologists have warned against scratching the face to relieve itching, because of the possibility of scarring. Instead of this the patient constantly hits

her head for days, weeks, and months. Given an individual with a susceptibility toward detachment, this could be an effective cause. Education of the patient by the dermatologist on this point may prevent some cases of retinal detachment.

Obstetricians

Congenital anomalies. Keep in mind the possibilty that congenital anomalies of the eye may develop if the mother sustains even a seemingly slight illness in the first trimester of pregnancy.

Dr. Theodore H. Ingalls, associate professor of epidemiology at the Harvard School of Public Health, cited recent experiments in which pregnant laboratory mice were subjected to the deprivation of oxygen before bearing their offspring. For five hours the mice were put in a vacuum chamber with an air pressure about the same as at the top of Mt. Everest.

The mice produced a large percentage of defective offspring, the defects varying according to the stage of pregnancy during which the mother was deprived of oxygen. If pregnancy had advanced to about 16 days before the deprivation of oxygen, a curious defect of the eye and eyelids, so-called "open eye," was produced. Ingalls in a personal communication defined the latter as "a complex deformity with many associated intraocular changes, some of which resemble those seen in retrolental fibroplasia."

The eye is most likely to be affected by noxious agents acting during the first few weeks of pregnancy, since the greatest interference with differentiation of that structure may be anticipated at that time. Minor febrile illness of the pregnant mother during the first month of pregnancy is most likely to effect anophthalmia, microphthalmos, and congenital cystic eye, according to Falls.[13] Infections contracted during the second month are more likely to produce congenital cataract and disturbance of retinal differentiation. Noxious agents such as rubella and other exanthems are known to pass the barrier of the placenta and affect the eye in this critical period.

What can the obstetrician advise his patient to lessen her possibilities of developing such infections at this critical time? It would seem to me that enlightening the mother as to keeping out of crowds, resting and building reserve, can be done without making the mother develop anxiety.

Every women, when her pregnancy is first diagnosed or recognized, should from that time on be under the constant care of her obstetrician. Public education should be undertaken so that every pregnant woman will be encouraged to keep a daily record during her entire pregnancy of any deviation from her normal routine health such as colds, abdominal disturbance, gastrointestinal upset, and the like.

An accumulation of such records correlated with examination of the newborn in say 100,000 cases could be most valuable in interpreting environmental changes and congenital defects.

Young women who have not previously suffered from rubella should be deliberately exposed to infection before pregnancy. If exposed in first three months of pregnancy, convalescent serum might be tried. If the patient should develop German measles during this critical period, therapeutic abortion should be considered.

Prophylaxis in newborn. Insist on care of the eyes of the newborn by two—doctor and nurse—to avoid the occasional forgetting of this prophylactic measure in the prevention of ophthalmia neonatorum.

In a hospital with which I am connected, four cases of gonorrheal ophthalmia occurred during the first six months I was there. On investigation, it was found that one nurse was assigned to the eye care of the newborn. Needless to say, it is not always possible for one person to separate the lids and instill silver nitrate in the eyes of a newborn child. Then there is always the possibility of this

important procedure being overlooked. The medical board passed a ruling requiring a doctor and a nurse to carry out this prophylaxis. There has been no further case in the past 10 years.

Congenitally obstructed tear ducts can frequently be diagnosed in the first few days of life by noting if there is undue reaction in the lids of one eye after the instillation of the silver nitrate in both eyes. In the normal eye it is carried off down the duct. On the obstructed side, where it cannot be carried off, it will give more swelling. This should be noted on the chart so that in the follow-up outside of the hospital, earlier diagnoses of obstructed ducts may be made.

Syphilis. Treat lues in the early stages of pregnancy; 2.8 percent of blindness in children is caused by prenatal syphilis. Prevent interstitial keratitis, which causes 1.2 percent of blindness in children.

Premature infants. See that every premature infant weighing four pounds or less at birth be under observation by an ophthalmologist; 15 percent of such babies will develop retrolental fibroplasia.

Foote and Johnson[14] believe that "if treatment is found for retrolental fibroplasia, it undoubtedly will be most effective when given early."

John C. Locke has concluded: "If the fundus is normal at the age of three months retrolental fibroplasia will not occur later. Cases are usually observed from eight days to eight weeks; 70 percent of those affected will end up with useful vision and only 30 percent will go blind."

Szewczyk[15] believes the process can be reversed by oxygenation (60 to 70 percent) in incubators and gradual weaning from the oxygen; whereas, Campbell[16] believes prophylaxis consists of (1) avoidance of intensive oxygen therapy by giving oxygen only when the infant is cyanosed and not as a prophylactic measure, (2) carrying the fetus to 33 weeks' gestation, (3) avoidance of overheating, (4) prevention of infection, and, should it occur, its prompt and efficient treatment. The final answer has yet to be given.

Pediatricians

Squint. See that squint cases are cared for as early as possible. Do not let the child "outgrow" the squint and end up with a useless eye.

Measles. Most scars in the cornea after measles are due to secondary invaders. In this disease, the cornea is studded with many punctate epithelial lesions. These produce the photophobia and tearing of the eye. Darken the room and caution against rubbing the eyes. With the use of antibiotics locally, when indicated, many of the ulcers with ensuing scarring can be prevented.

Ophthalmologists

1. *Glaucoma prevention.* As his contribution toward preventing glaucoma, the ophthalmologist should:

Do routine tonometry on all patients over the age of 30 years unless the eye is definitely soft to digital palpation. Once a diagnosis of glaucoma is established, instruct the patient in the necessity of being observed at regular intervals.

Emulate the fine start made by Brav and Kirber[17] for the Philadelphia Committee for the Prevention of Blindness. They screened 10,000 persons between the ages of 40 and 65 years and found 1.53 percent had undiscovered glaucoma; including borderline cases, two percent of the general population were shown to have glaucoma.

If we are going to try to detect symptomless glaucoma cases, we must educate the optometrists, who number 1,000 in the five boroughs of New York City and who see about seven out of every 10 who have their eyes refracted.

I suggest that the Eye Section arrange a list of willing lecturers among its membership and work out a program that would allow one lecture a month to optometrists to keep the subject fresh in their minds at all

times. All doctors desirous of participating would be given a chance and a part in the plan.

2. *Teach your patients*—"If good vision you do prize, keep your fingers from your eyes." Ocular infections may be transmitted by finger-to-eye contamination.

a. At the time when all New Yorkers were vaccinated because of a smallpox scare, everyone saw cases of vaccinia of the eye—lid, conjunctiva, or cornea—transmitted by careless touching of the eye after either handling gauze that had been on a vaccination or after touching the vaccination itself.

b. In this country, practically every case of gonorrheal conjunctivitis in adults appears after finger-to-eye contact. In Egypt primary gonorrheal conjunctivitis occurs, but here it is secondary to urethritis usually.

c. In epidemic keratoconjunctivitis the method of spreading may be the same. Thygeson, in a personal communication, stated, "in 22 of the 23 doctors that I know who have contracted the disease, finger-to-eye transmission was the cause. In the 23rd, irrigation fluid was accidentally splashed into the eye while the physician was working on a case."

d. Women who carry the virus of inclusion-body conjunctivitis in their genital tract have infected their eyes by finger-to-eye contact as no general immunity is developed.

3. *Molluscum contagiosum and warts* at the edge of the lid should be removed to prevent possible conjunctivitis and keratitis.

Molluscum contagiosum vary in size from a pinhead to a hazelnut. They contain the Henderson-Patterson inclusion bodies. If they occur at the edge of the lid, they may produce not only a follicular conjunctivitis but corneal vascularization so severe as to resemble trachoma. The tumors may be hidden by the eyelashes or masquerade as desquamated epithelium. The corneal lesions are caused by toxic substances diffusing from the nodules. Removal of the nodule clears up the eye condition and will prevent the eye condition if removed early enough.

de Roetth described 11 patients in whom subacute conjunctivitis and severe superficial keratitis or corneal ulcer were associated with the presence of warts along the lid margin. Removal of the warts relieves the condition. One should therefore look for such lesions, routinely.

4. *Industrial ophthalmology.*

a. Insist on goggles to prevent intraocular and corneal foreign bodies. No community goggles allowed.

b. Make X-ray films of all potential cases of intraocular foreign bodies.

c. All cases of mature unilateral cataract should be studied by X ray when the cause is undetermined; occasionally a foreign body may be found.

d. Dilate all eyes after injury to rule out retinal detachment, particularly if a vitreous hemorrhage is present.

5. *Ocular Surgery.*

We might consider the following measures as bearing on preventive ophthalmology:

a. Prophylactically—do lens sensitivity testing in milky white cataracts in allergic patients.

b. Always have proper anesthesia and lid akinesia before intraocular surgery.

c. Decompress very slowly in glaucoma cases.

d. Clear up infected sacs before intraocular surgery.

e. Be sure the conjunctiva is clean before eye surgery.

f. Trim lashes, if you do, before the patient is brought to the operating room to prevent cilia from getting into the anterior chamber.

g. Do not put ointment into the cul-de-sac at the time of intraocular surgery.

h. Never hold sutures in removing them after cataract surgery. Cut them first and then lift them out.

i. Masks should be worn over the nose, not under it.

j. Never operate if you have an acute respiratory infection.

k. One should learn to operate with gloves.

l. No one should be allowed in the operating room with street clothes on.

m. No electric fans should be permitted in the operating room.

n. Nurses should change gloves after preparing the patient.

o. Individual wash bottles should be used.

p. Individual dropper bottles should be supplied for each patient.

q. Dip all instruments that enter the anterior chamber before using a second time.

r. The problem of airborne lint and airborne infection in ophthalmic surgery from the preventive aspect has been emphasized by Post:[18] (1) Covers should be kept on all solutions and instruments as much as possible; (2) treat all blankets and drapes with oil solution; (3) treat operating room floor with a dust-allaying preparation.

s. Vail[19] suggests that pieces of rubber sometimes seen in the anterior chamber, originate in the irrigator. This complication can be avoided by using a syringe with a metal irrigating tip. Smith[20] cautions against the use of glass tips. One should never use dry cotton around the eye in intraocular surgery.

Chamlin[21] warns against talcum from rubber gloves getting into the operative field particularly in muscle surgery. This can be avoided by washing gloves with sterile saline after putting on and using biosorb instead of ordinary powder.

t. A word about the preoperative use of penicillin both locally and systemically. First of all, this antibiotic only holds in check gram-positive rods. If coliform or pyocyaneus bacillii are present, they will grow much more rapidly after the removal of the gram-positive organisms. Secondly, if the patient happens to be allergic in type, an anaphylactoid reaction may occur and the surgeon will be startled by a markedly chemotic eye at the first postoperative dressing. Once the anterior chamber has reformed, doses of 300,000 u. of penicillin daily get into the anterior chamber in such negligible quantities as to be of little value. For these reasons, if medicaments are used preoperatively, chloromycetin or gantrisin would seem to be the agent of choice.

6. *Contagious diseases.* In the 1941-42 spread of epidemic keratoconjunctivitis, office transmission was rife, many cases were spread by tonometers or contaminated pontocaine. In one hospital in a glaucoma clinic, a patient came in with an unrecognized epidemic keratoconjunctivitis. Within 10 days, eight patients who had had their tensions taken after this case were all infected with the disease.

Thygeson[22] suggested the following to help restrain the spread of this disease: (1) Be familiar with the disease; (2) wash your hands between examinations; (3) see epidemic keratoconjunctivitis cases apart from other cases, protect against contamination of door knobs and treatment chairs; (4) discourage finger-to-eye transmission; (5) do not spread infection by your tonometers (sterilize them); (5) discard dropper bottles and use individual sterilizable droppers.

7. *Sterilization of solutions.* Be sure all eyedrops in your office are made up with antiseptics. For fluorescein use sodium ethyl mercuri thiosalicylate (1:7,500). For other medicaments not antiseptic in themselves, cetyl-dimethyl benzyl ammonium chloride (1:10,000) is advisable. Contamination with Pseudomonas aeruginosa has occurred in fluorescein, eserine, cortisone, penicillin, and some sulfa preparations.

In testing Pseudomonas aeruginosa for agents that would inhibit it, Carbonaro used the phenol coefficient method described by Gradwohl. Two strains of Pseudomonas aeruginosa recovered from corneal ulcerations occurring in patients were used. She found the following drugs of value in vitro as bactericidal agents:

Sulfamylon (5.0 precent), zephiran (1:5,000), polymyxin (0.25 percent), cetyl-dimethyl benzyl ammonium chloride (1:10,000), gantrisin (4.0 percent), streptomycin (10,000 units per cc.).

The following were of no value: Chlorbutanol (0.5 percent), terramycin (25 mg. in 5.0 cc.), furacin ophthalmic liquid, sodium sulfacetamide (30 percent), chloromycetin (25 mg. in 5.0 cc.), sodium propionate (5.0 percent).

Sanacore, at my suggestion, asked 22 of the leading drug firms these three questions: (1) Are your eye preparations sterile? (2) What means do you use to obtain sterility? (3) Do you test each batch issued for sterility?

All answered, but only five of the 22 companies that manufacture eyedrops and ointments said that their solutions were sterile, checked for sterility, and gave the method of sterilization. One company stated that "no ophthalmic ointment can be absolutely sterile as any method of sterilization after the material is fixed and filled into tubes would cause a separation of the contents." Others hid behind "existing Federal regulations do not require sterility of ophthalmic preparations" and "official correspondence and ophthalmic formularies do not emphasize absolute sterility." The situation speaks for itself and demands:

a. Passage of the necessary legislation to insure sterility. Minsky and Theodore have taken a step in this direction by a letter to the editor of the *Journal of the American Medical Association.*

b. Get the drug stores to fill prescriptions in sterile fashion. There are some medicaments put up sterile, but the druggist uses nonsterile graduates to measure off the diluent, hence the break in the technique.

8. *Etiologic diagnosis.* With specific medication for gram-negative and gram-positive organisms, exact etiologic diagnosis is imperative. A pyocyaneus ulcer of the cornea can be stopped short if streptomycin in adequate strength is used frequently and early enough. If it is treated with penicillin, the eye will be lost.

Likewise a petit ulcer of the cornea stands a better chance of cure if zinc and the sulfonamides are used than if penicillin, silver, or other medicaments, useless for this condition, are employed. One cannot be sure of the etiologic diagnosis clinically but laboratory information makes the task simpler.

9. *Heat and light rays.* Be aware that ultraviolet and heat rays may cause macular lesions in the aphakic eye. Use tinted lenses in all glasses prescribed for cataract patients. The cases that may develop macular lesions can be recognized by the excessive photophobia and lacrimation noted at each dressing.

10. *Venous occlusion.* Recognize the danger signals of retinal venous occlusion as suggested by Bertha A. Klien.[24] Subjective symptoms are intermittent visual obscuration with no apparent reason. Object findings are:

a. Engorgement of the venous tree and edema of the corresponding sections of the optic papilla and/or the retina along the involved veins. Careful comparison of both eyegrounds is imperative.

b. Unilateral absence of physiologic venous pulsation in the involved eye or a difference in the ease with which the venous pulsation can be produced (where none exists) in the two eyes.

c. Dilation or new formation of tortuous and thin-walled vessels in one or more places at or near the disc. These point to an early attempt of collateral circulation.

d. Multiple crossing of the same vein or congenital loops or twists.

e. A combination of therapeutic agents, such as penicillin and digitalis, which creates conditions favoring a thrombus.

Case report. A-54-year-old man had essential hypertension. Visual obscurations had occurred eight months ago. Edema was present along the superotemporal vein, peripheral to arteriovenous crossing. There was no venous pulsation (although this was marked in opposite eye). After six months on dicu-

marol, intermittent obscuration disappeared. Neither edema nor venous pulsation returned.

Discussion

Fifty percent of blindness in children is due to congenital and hereditary causes. What can one do to prevent such hereditary diseases as retinitis pigmentosa, Leber's disease (hereditary optic atrophy), cerebromacular degeneration, amaurotic familial idiocy (Tay-Sacks disease), and juvenile amaurotic family idiocy?

Falls states: "The giving of genetic advice must consider:

"1. The aberration may have resulted from a mutation occurring in the germ plasm of the two parents.

"2. The second possibility is that the aberrant trait may have been the result of a recessive mode of transmission. Since related individuals are more likely to possess similar germ plasm, the question of consanguinity is most important in the diagnosis of recessive disease states."

Statistics suggest that, if one child is born with a hereditary pathologic condition of the eye, there is a 10 to 12 percent greater chance of there being another similarly affected offspring. Holst suggests that voluntary sterilization should be considered as a means of preventing hereditary blindness. He also suggests a ban on marriage of persons showing hereditary blindness as a dominant characteristic. The final answer cannot be given at this time. With the acquisition of more specific knowledge of the inheritance pattern of disease, prophylactic eugenics may, however, become practical.

Reports should be required for unilateral blindness (below 20/200). It should be noted how that particular case could have been prevented (if at all possible) in the opinion of the ophthalmologist or as suggested by the patient (if the patient's reason seems logical to the ophthalmologist).

Every eye removed and examined should be reported by the pathologist so that a better histopathologic classification of ophthalmic disease processes may be built up.

All of us can do much to prevent some eye disease if we utilize existing knowledge. Can we afford to do less?

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IMPORTANT COMPLICATIONS IN RETINAL DETACHMENT AND THEIR INFLUENCE ON THE SURGICAL RESULTS*

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Two years ago, at the first clinical conference of the Wills Eye Hospital, I presented the results of retinal detachment surgery in over 400 cases. Since that time I have done over 100 additional cases, making a total of over 500 cases. I feel that I have had my share of complications and am qualified to report on them.

Dunnington,[1] Koenig,[2] and others have written on the causes for failure and complications in retinal detachment surgery, and I am in full agreement with the complications as outlined by them. However, since my series of cases is unusually large for one individual, it is natural that I should have encountered a large number of complications which could be studied.

For the purpose of this discussion, the complications of retinal deatchment that influence the surgical results may be divided into the following four classifications:

1. Preoperative, or those which occur prior to surgery.
2. Those which happen at the time of surgery.
3. Those which develop after surgery.
4. Those which cannot be classified.

PREOPERATIVE COMPLICATIONS

1. An extensive hemorrhage may occur at the time of the tear. This may cause a heavy cloud in the vitreous which prevents a satisfactory view of the fundus, making it difficult to see the detachment and impossible to locate the tear.

2. Vitreous opacities due to a recent inflammatory process are also frequently associated with hypotony, the presence of which indicates a poor prognosis. However, without hypotony, if the lesion is in the periphery, the prognosis may not be too bad.

3. The failure to find a tear may be a serious complication and have a definite effect on the outcome. I am sure though that I have closed peripheral tears that could not be seen and have obtained favorable results in many of these cases. The tear should be looked for repeatedly with the giant ophthalmoscope and good illumination.

Placing the patient in a favorable position in bed to allow the force of gravity to encourage the retina to settle back in place makes it possible to observe tears not seen previously. Indirect ophthalmoscopy has been advised by Schepens[3] and others. This may be beneficial in detecting a peripheral tear or small disinsertion but, in my experience, the details are better seen by direct ophthalmoscopy.

4. Detachments of the retina that do not settle back in place after a reasonable length of time, when the patient has been quiet and in a favorable position with goggles or bandages, always offer a poor prognosis.

5. A very extensive tear, more than one tear in different areas, tears involving the macula, or tears far back and near the optic disc are all complications that make the prognosis for surgery less favorable.

6. When the detachment is extensive and involves the macula, central vision is always poor and the prognosis should be guarded. Children and young adults seem to tolerate macular involvement longer than older patients.

7. When the detachment has been present for too long a period of time before surgery, the prognosis is poorer but not hopeless: One is often surprised at the favorable results in such cases.

8. Detachments associated with fibrous bands, hemorrhages, and newly formed vessels, as seen in diabetes, always present a serious problem and offer a poor prognosis. Also,

* Read before the third clinical conference of the Wills Eye Hospital in Philadelphia, March, 1951.

when they are associated with fixed folds in the retina and a retraction of the vitreous as described by Schepens,[3a] the prognosis is very poor.

9. The possibility of a tumor should always be suspected. This diagnosis should be made by the appearance of the detachment which is of slate color, smooth, dome-shaped and circumscribed with no tear visible. This picture is not always definite and the diagnosis is not easy or certain. Transillumination, which may or may not be interfered with, is questionable and at times misleading. Visual fields should be studied with full and reduced illumination. These may show a smaller absolute scotoma to full illumination corresponding to the area of the tumor inside a larger scotoma to reduced illumination. This latter scotoma corresponds to the serous portion of the detachment.

A posterior sclerotomy with a search for tumor cells may help. If these are found, a positive diagnosis of tumor can be made but, if not found, it does not necessarily indicate a negative diagnosis.

Sometimes a diagnosis of tumor is not possible before operation. After surgery the serous part of the detachment may recede and leave the solid part more prominent, thereby making the diagnosis easier. This happened in one of my cases in which the eye was enucleated a few days after the detachment surgery and the diagnosis of a tumor was confirmed.

10. Sometimes it is difficult to dilate the pupil satisfactorily. It may be necessary to use an injection of atropine, adrenalin, and cocaine subconjunctivally. The patient may become sensitive to atropine and then scopolamine (0.25 percent), neosynephrine (10 percent), or some other mydriatic may have to be used. More external or scleral pressure by various instruments may be necessary in these cases to bring the periphery of the retina into view.

11. A retinal detachment may be secondary to intraocular foreign bodies. The foreign body should be removed first and the detachment repaired as soon after this as possible. It is not uncommon to have contracting bands of fibrous tissue or hemorrhage in the vitreous complicating these cases.

12. Aphakia, per se, may be a complication, but in my experience it has not been as serious as reported by others. There are certain complications, which may occur at the time of/or following the cataract surgery, which lead to retinal detachment and reduce the chances for a favorable result in retinal detachment surgery.

These are: (1) An unusual amount of vitreous loss; (2) a large vitreous hemorrhage with organizing bands; (3) a prolonged postoperative iridocyclitis; (4) secondary capsular membranes or a thickened hyaloid, with a pulled-up iris, so that a satisfactory ophthalmoscopic examination or search for and localization of the retinal tear is impossible; (5) the condition of the vitreous which may be detached with retraction and contraction bands attached to the retina.

This last condition was stressed by Lindner[4] and recently Schepens[3a] called attention to this complication and explained its mechanism somewhat more in detail. Of course, this condition or an early detachment of the retina may precede the cataract surgery and be undetected.

Naturally, all of these complications make the prognosis poor. I have experienced approximately 50-percent success in my cases of aphakia and I am sure that the absence of the conditions just mentioned was largely responsible for this relatively good percentage of favorable results in this type of case.

Complications at time of surgery

1. Perforation of the sclera with the needle at the time of the retrobulbar injection can occur as it did in one of our cases. No serious results followed, however, since the mistake was recognized before the anesthetic was injected. This is more likely to occur in large myopic eyes with thin scleras.

2. Retrobulbar hemorrhage may occur at the time of the injection of the anesthetic,

which may make further operative procedures very difficult or impossible.

3. Perforation of the sclera with scissors may happen at the time of the dissection for its exposure. This is more likely in cases which have been previously operated, where the sclera has been perforated with numerous micropins and is dried out and quite friable.

4. Tearing of, or injury, to the vortex veins may cause annoying hemorrhage externally, or intraocular hemorrhage which may be more serious.

5. Too much current with the first few needles may cause loss of fluid, giving rise to a wet field and hypotony, making it very difficult to insert more needles. For this reason the current of the diathermy machine should be kept on the low side, 50 to 60 ma. to start with, so that the micropins go in slowly and remain firmly fixed in the sclera. The field should be kept dry and extreme care should be taken not to dislodge any of the micropins.

6. Drying of or injury to the corneal epithelium may cause some edema or opacification which interferes with the ophthalmoscopic examination of the retina and prevents a clear view of the tear.

To avoid this, the conjunctival incision is made as far away from the limbus as possible in order to obtain a large conjunctival flap which can be moved back and forth by traction sutures, two or three of which are placed in the anterior cut edge of the conjunctiva. These are all twisted and held together by a serafin clamp. This protects the cornea, keeps it clear, and permits frequent observations of the fundus with the ophthalmoscope. It also enables one to control more accurately the closure of the retinal tear.

7. Intraocular hemorrhage may be caused by injury of the choroidal or retinal vessels with the micropins, by injury when trephining the sclera, or when perforating the choroid with the gradual lacrimal dilator.

8. Too extensive coagulation with shrinkage and burning of the sclera may give rise to an hourglass contraction of the sclera, which might prevent the approximation of the retina to the choroid. Also, too much coagulation may cause injury to the ciliary body with hemorrhage and exudate in the anterior chamber, which sometimes is seen at the end of the operation or at the first dressing. This always leads to an iridocyclitis of some degree, and in at least six of my cases it caused phthisis with complete loss of vision. However, in only one case was it necessary to remove the eye.

9. Macular damage by coagulation or hemorrhage. The first of these should be avoided by watching for landmarks and checking ophthalmoscopically. One should remember that the macula lies very close to the anterior or superior portion of the insertion of the inferior oblique muscle, just 29.2 mm. back of the limbus in line with the superior edge of the external rectus muscle. One should never use long pins or deep coagulation in the macular region; only superficial or surface coagulations should be used here and these only when absolutely necessary.

Hemorrhage sometimes occurs near the macula and may seep into the macular region. Occasionally this may be checked, if discovered soon enough, by changing the position of the patient so as to encourage the hemorrhage to flow or settle by gravity away from the macula.

10. Failure to coagulate the choroid occurs sometimes because of atrophy. Arruga[5] states that this occurs in detachments with large tears or disinsertions where the vitreous has been in contact with the choroid for a long period of time. I have seen this and I am sure that it accounts for at least seven of my failures. In these cases one must place the areas of coagulation farther back into healthy choroid and be content to prevent extension of the detachment, with a smaller visual field rather than no field.

11. Failure of the retina to go back in place due to inadequate drainage of the subretinal fluid. This complication may be avoided by making more than one opening in

different portions of the sclera with the trephine or scissors. Then when the choroid is exposed through the scleral opening it is perforated through into the subretinal space with a blunt-tipped gradual lacrimal dilator. This allows free drainage of the subretinal fluid, which may be further encouraged if necessary by using suction with a medicine dropper over the micropin perforations.

12. If the retina still does not go back in place after the procedures just described have been followed, the injection of air or normal saline into the vitreous cavity may be considered.

This is done through the pars planum in phakic cases by means of an incision with a knife needle through the sclera and then the insertion of a lacrimal cannula. Another method is to use a hypodermic needle and pass this through the sclera by means of the application of the diathermy electrode and current.

In aphakic cases the cannula is passed through the pupillary area after making a beveled incision in the periphery of the cornea with a knife needle. A two-cc. hypodermic syringe filled with air is attached to the lacrimal cannula in each case.

Postoperative complications

1. Unusually marked reaction may be present at the first dressing. This may result from cutting too many muscles or from pulling too hard on some muscles that should have been cut. It may also result from an excessive amount of electrocoagulation and dissection. Of course, infection may also be a cause for such a marked reaction. However, we have never had any serious residual effects from this.

2. A hazy cornea may be due to too much coagulation or to a corneal abrasion at the time of operation, as mentioned. Occasionally serous detachment of the retina may be associated with chronic simple, or wide-angle, glaucoma.

At least two eyes in my series had this condition, associated with the detachment, before operation. As a result the corneas were somewhat hazy and I was unable to dilate the pupils as widely as I would have liked. Naturally, tumor must be thought of in these cases. Injection of the vitreous with air or other substances can also cause an elevated tension with a hazy cornea, which may persist after surgery.

3. The anterior chamber may be filled with an exudate and/or hemorrhage and the vitreous become very cloudy, due to an iridocyclitis. This is usually due to the use of too much diathermy current at the time of surgery. Sometimes too much damage is done to the ciliary body and gives rise to this condition in the anterior chamber.

4. Sometimes vitreous, retinal, or choroidal hemorrhages may be due to vomiting or excessive movements of the patient's head and eyes too soon after surgery.

5. Lens changes, or partial cataract, may take place and cause a marked reduction in vision in cases that are otherwise successful. These usually occur six months to three years later and are apparently due to too much diathermy current. This should be guarded against.

In my series, approximately five percent of the cases developed cataracts which we felt were due to excessive diathermy current. Most of these developed in the operated eye of young adults who had a clear lens in the other eye.

The opacities all started in the posterior cortex in the form of a bubble or two and these gradually multiplied, causing a considerable opacification with some reduction of vision, but to date none has matured sufficiently to require extraction.

6. Infection of the conjunctival wound sometimes occurs. This may result from an infected conjunctival sac or a contaminated suture. Tenonitis can develop and may be very serious. We had two such cases which were fairly well localized and were relieved by incision and drainage, with no serious effect on the final result.

7. Infection of the temporarily tenotomized

muscle stump about the sutures, associated with granulation, was quite common when catgut sutures were used. Cotton sutures, which we have been using recently, cause less reaction and granulation. The granulation, which usually occurs some weeks after operation, should be excised with the suture knot. This, with the use of an astringent eye wash, usually relieves the condition promptly.

8. Redetachment of the retina not infrequently occurs when the patient gets up. This, of course, is due to the fact that the coagulations have not made contact with the retina or have not been in close enough contact for a long enough time. The patient should be elevated very gradually, especially when he has been in bed with the head low and the feet up.

9. Extensive hemorrhage into the vitreous may occur when the patient gets up too fast. The absorption of this is sometimes hastened by fever therapy and potassium iodide internally. The outcome is sometimes better in these cases than one would expect, although visual recovery is always delayed.

10. Cystic degeneration of the macular area, as described by Reese,[6] often appears after operation, due to the fact that the macula was involved by the detachment for too long a time before operation. Central vision is always poor in these cases, especially in elderly people, where the retina has been detached in the macular area for longer than a few days.

11. Vitreous opacities, of some degree as a rule, are present in all cases and sometimes these may be severe. Most of these gradually absorb in the course of time. Iodides are given in the form of Organidin for a period of months postoperatively. It is surprising how many patients can take iodides in this form for a long time. If the opacities are severe, fever therapy should be given before the patient leaves the hospital.

12. Proliferating retinitis with bands sometimes follows a large vitreous hemorrhage. Also retraction of the vitreous may follow surgery. The prognosis is always bad when either of these conditions is present.

13. Filamentous keratitis has not been mentioned very often in the literature as a complication of retinal detachment surgery. It was found to be a not infrequent complication and occurred in six percent of my cases. It was present just as often in the unoperated eye as in the operated eye. It is felt that the long period of bandaging of both eyes has a definite influence on the development of this condition, and, for this reason, it is preferable before operation to use pinhole goggles during the day rather than continuous bandages.

In every case in which this complication occurred, relief was obtained by cauterization of the loose areas of epithelium with 20-percent trichloracetic acid. The areas cleared immediately and remained clear after the patches were removed and the goggles were again worn.

14. Ptosis of the upper lid, due to improper closure of the conjunctival wound, may occur. This occurred in one of my cases when the superior margin of the tarsus with the levator muscle was caught and pulled down to the globe.

15. Phthisis bulbi may occur later, and sometimes does, with irritation sufficient to require enucleation. This happened in one case previously mentioned.

16. Too much freedom and movement of the eyes too early may ruin what would otherwise have been a good result. This must be controlled by a definite routine of gradually increasing the patient's ocular movements by means of goggles, the openings of which are gradually enlarged. Later, the patient's refractive error can be corrected and glasses ordered in the form of modified goggles, which help to carry out this principle for a longer time and at the same time allow the patient to get back to some type of work.

Phorias or tropias occasionally occurred, but in no case did this persist to such a degree that muscle surgery was necessary. When it did occur, it was due to a shortening or strengthening of the temporarily tenotomized

muscle, rather than a weakening of this muscle.

Systemic complications such as backache, nausea, and vomiting should be controlled before operation, but sometimes may occur after operation. These should be treated symptomatically.

In spite of the advanced age of many of my patients—and, contrary to what one would expect—only one case of pneumonia occurred in my entire series. It is also interesting that this occurred in a patient who was sitting up in bed the entire time, since his detachment was below. The pneumonia was treated and relieved with aureomycin and this complication did not interfere with a good visual result.

Femoral thrombosis occurred in one patient who had previously suffered from a pyonephrosis. This condition could not be controlled and resulted in the only death experienced in the entire series. There were two other cases of pulmonary thrombosis which responded favorably to treatment.

Unclassified

Bilaterality might be mentioned here. Some people appear to be born with the retinas in both eyes predisposed to detachment. Twenty percent of the spontaneous detachments in my series were bilateral and I am sure that the percentage will be higher as time is given for the second eye to become involved. This usually occurs five to 10 years after involvement of the first eye and usually a corresponding area of the second eye is involved with relatively the same type of tear.

The prognosis in these cases is always bad. When the detachment is not accounted for by trauma, hemorrhage, inflammation, or other known causes, electrocoagulation surgery should always be advised with the hope of saving even a small amount of vision in the eye because of the possibility of the other eye developing the same condition. From my own experience I am convinced that spontaneous retinal detachment is a bilateral condition.

Finally, there are some cases that, from every point of view, it would appear should obtain a satisfactory result but, for some unknown reason, they just do not.

Summary

The complications of retinal detachment and their influence on the results of surgery have been given. Based on my experience in a large series of cases, the complications have been divided into four groups: (1) Those occurring prior to surgery, (2) at the time of surgery, (3) after surgery; and (4) unclassified.

Conclusions

After reviewing this series of over 500 cases and listing the various complications that occurred, one is impressed with the percentage of good results obtained from surgery. I feel that I have learned a great deal from my mistakes and failures. Furthermore, it is my hope that this analysis of the complications in a series of cases, such as presented, will help others in the future to avoid many of my mistakes and be able to give a better and more accurate prognosis for individual cases as they present themselves.

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ORBITAL EMPHYSEMA

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In 1943, I presented in a previous paper on orbital emphysema, a new method worked out with the coöperation of Dr. Paulo Filho, an ophthalmologist, and Dr. G. Ribeiro, my assistant.

A 10-year-old girl, presenting exophthalmos of the left eye, attributed to an orbital tumor, was sent to me for radiotherapy. Since it seemed necessary to confirm the diagnosis and to know the tumor situation, it occurred to me to induce orbital emphysema by injecting air into the cellular

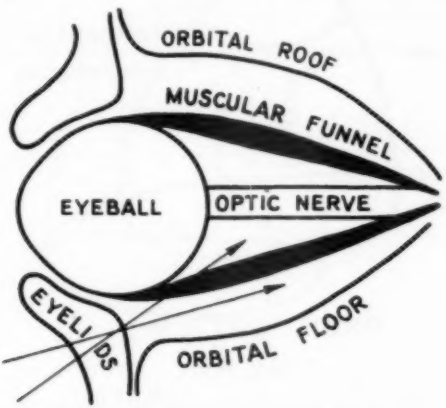

Fig. 1 (de Abreu). Diagram showing technique of injecting air.

adiposus space, inside and outside the muscular funnel of the eye. The artificial emphysema would show the form and position of the eyeball and the existing orbital tumor, as well as the relation between them.

It is well known that the orbital cavity is not accessible to X-ray examination. Radiography or tomography do not show the intraorbital contour of the eyeball and the orbital contents. The normal and pathologic tissues have practically the same density and cannot be separated from one another.

Fig. 2 (de Abreu). Lateral radiograph. Superposition of the bone effaces the emphysema and the soft tissues of the orbit.

A few days later, with the coöperation of Dr. Paulo Filho, emphysema was induced by the injection of about eight cc. of air into

Fig. 3 (de Abreu). Lateral tomography through the center of the eyeball produces a sharp contour of eyeball and tumor. The tumor resembles a dense semicircle behind the eyeball and adherent to the orbital roof.

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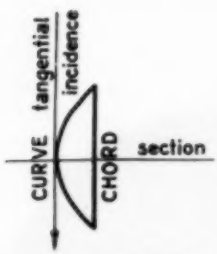
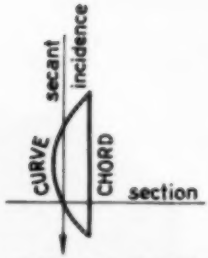


CENTRAL SECTION

Fig. 4



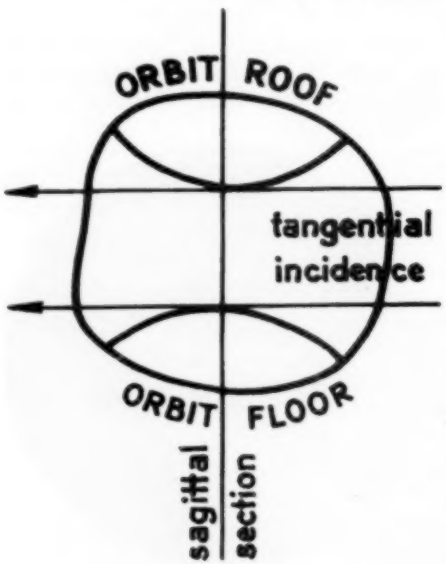
ECCENTRIC SECTION

Fig. 5

Fig. 4 (de Abreu). The tomographic section through the eyeball or tumor curve should be perpendicular to the middle of the corresponding chord. Under these conditions the normal ray is tangential to the curve of contrast, which has a sharp limit.

Fig. 5 (de Abreu). If the tomographic section is not median, the normal ray intercepts the curve as a secant and projects a blurred image.

the retrobulbar space. The technique is quite similar to that used by ophthalmologists in deep anesthesia of the ciliary nerves (fig. 1).

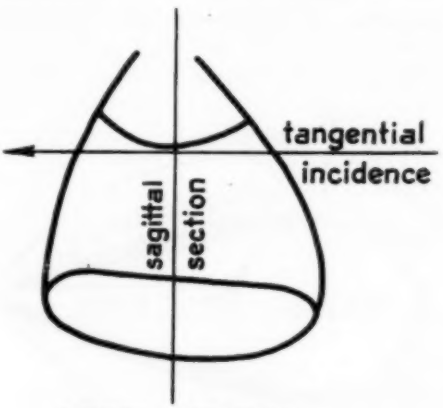


ROOF AND FLOOR TUMORS

Fig. 6 (de Abreu). Tumors situated on the roof or floor of the orbit require lateral tomography.

It is important to be sure that the needle is not inside the orbital vessels, to prevent gas embolism. The needle course must avoid the region probably occupied by the tumor, opposed to the eyeball displacement; then the air should be slowly introduced with a syringe.

The artificial emphysema swells the tissues. The insufflation causes a slight pain, which is tolerable and does not require the use of analgesics. After the X-ray picture has been taken, a subcutaneous puncture and



ORBIT BOTTOM TUMORS

Fig. 7 (de Abreu). Tumors situated on the bottom of the orbit are visible with lateral tomography.

aspiration remove the air; the eye turns back to its initial position and the signs of emphysema disappear.

The radiologic technique is very important, because the emphysema produces slight contrast and the bones are superposed to the soft tissues (fig. 2). According to my experience, radiography is not to be recommended. Only tomography eliminates the bone superposition and shows clearly the emphysema transparency. So, without the use of tomography, orbital emphysema cannot be performed.

In the case of exophthalmos herein presented, median sagittal tomography demon-

strates clearly the eyeball and the tumor shadow, which are surrounded by the emphysema. The tumor appears as a dense semicircle situated behind the eyeball and adherent to the orbital roof (fig. 3).

How clear the X-ray picture is depends on the direction of the rays on the opposition surfaces. I must explain that the opposition surface produced by the contrast between the air and the soft tissues is situated in the section space. Therefore, the tomographic cut through the eyeball or tumor curve

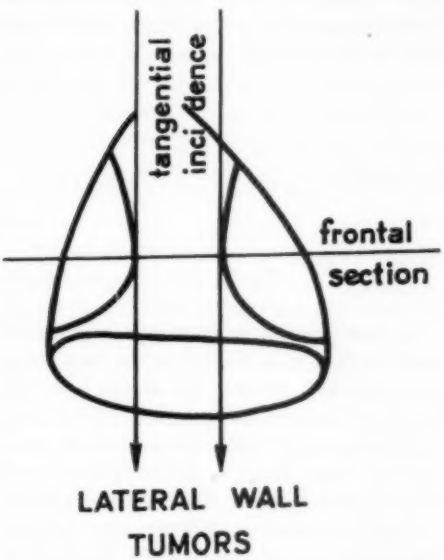


Fig. 8 (de Abreu). Frontal tomography demonstrates tumors on the lateral walls of the orbit.

should be perpendicular to the middle of the corresponding chord (fig. 4) so that the normal ray will be tangential to the opposition surface and project a sharp limit. If the section does not intercept the center of the chord (eccentric section), the ray will be secant and blur the shadow limit (fig. 5).

Based on these fundamental principles, the sharp limit can be seen on the axial sections, which intercept the eyeball center, in the frontal, lateral, and oblique planes.

Tumors on the roof, floor, or bottom of the orbit must be examined by lateral

Fig. 9 (de Abreu). Orbital emphysema. Frontal tomography, with the section intercepting the eyeball center and producing a sharp contour of the eyeball.

tomography (figs. 6 and 7); frontal tomography demonstrates well tumors of the lateral walls (fig. 8).

Three frontal and three lateral views are recommended. I now perform simultaneous tomographies by which three views can be taken simultaneously on the same film.

On the frontal plane (figs. 9 and 10)

Fig. 10 (de Abreu). Frontal tomography. The section is situated one cm. behind the center of the eyeball, which presents a blurred appearance.

tomographies may be taken in two positions: occipitofrontal and occipitomental; sometimes a slightly oblique position will show the tumor limit more clearly.

Several orbital emphysemas have now been performed without any difficulty. The apparent presence of an orbital tumor requires an accurate diagnosis in order to make a decision as to whether surgery or radiotherapy is indicated. Generally, orbital emphysema gives proof as to the presence of a tumor.

In eight cases clinically suspected of having orbital tumor, the new method showed the tumor shadow. Topographic locations of the growths were: five on the orbital roof, one on the roof and at the bottom, two on the internal wall.

Operations were performed on four patients and the emphysema diagnosis was confirmed (sarcoma, chondrosarcoma, osteosarcoma, and carcinoma).

If there is a tumor shadow, tomography shows its size, configuration, and topography. The differential diagnosis between several pathologic conditions may be very difficult (subperiosteal hematoma, cysts, vascular tumor, malignant tumor). However, the information given by orbital emphysema seems to be very important.

OPTIC ATROPHY IN INFANCY, CHILDHOOD, AND ADOLESCENCE*

A SURVEY OF 81 CASES

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Optic atrophy is a not uncommon affliction in infancy, childhood, and adolescence. In 1928, Lamb[1] surveyed the 599 pupils of the Missouri School for the Blind and found that 84, or 15 percent had lost their sight as a result of optic atrophy and that it had been the largest single cause of blindness (in 223 cases, or 17.5 percent) among the 1,272 pupils that had attended the school over a period of 15 years. Bishop Harman[2] reported optic atrophy in 222 cases, or 20 percent, of the 1,100 children in the London School for the Blind; 125 of these had congenital syphilis. A much higher incidence was reported by Masters,[3] who found that of the 187 blind children at the Indiana State School for the Blind, 61, or roughly 34 percent, were blind as a result of optic atrophy.

It should be borne in mind that since all these studies were limited to children living and attending school, the findings do not represent the total incidence of optic atrophy in children which would also include all cases arising from tumors, hydrocephalus, and other causes often leading to early death.

Optic atrophy in children may be produced by a variety of conditions. As the basis for an examination of the causes of the disease in the 81 cases observed at the University of California Hospital in San Francisco in the past 30 years, a review of the various factors known to have been responsible for the condition seemed to be in order.

DIAGNOSIS

The diagnosis of optic atrophy in children may be difficult. The color of the disc varies a good deal with the individual and in infants is definitely paler than in adults. For the certain diagnosis of primary optic atrophy there must be (1) pallor of the disc, (2) recession of the disc, (3) lowered visual acuity, and (4)

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field changes. For example, patients who have suffered from encephalitis or disseminating sclerosis may have pallid discs and yet have normal vision and fields. In such cases the axis cylinders have been depressed during the acute disease but have not been destroyed. Thus pallor alone is no criterion for optic atrophy. While it is impossible to take vision and fields in infants and very young children, they can be taken, with patience, in children aged five or six years and older.

Beauvieux[4] calls attention to the fact that there may be a gray pseudoatrophy at birth due to slow myelinization. This process normally reaches the disc by birth, but it may be delayed and there may then be a pseudoatrophy until the myelinization is complete. Beauvieux stresses the importance of repeated examinations before diagnosis is made and warns against alarming parents prematurely.

One condition to be kept in mind in the diagnosing of optic atrophy in infants is aplasia of the nerve, or the more frequent but rarely seen partial aplasia or congenital hypoplasia. Complete aplasia of the optic nerve was observed by Krauss;[6] in the case reported by him there was a myopia of 14 diopters due to ectasia in the region of the entrance of the optic nerve but neither the papilla nor the vessel funnel was visible. Hypoplasia, or partial aplasia, has been reported by Scheie and Adler,[7] Jerome and Forester,[8] and others. It is seen in patients whose vision is very poor or entirely lacking. The fundi show very small, pale discs, which are sometimes cupped, and the central vessels are in evidence. The small discs serve to differentiate this condition from atrophy of the nerve in which normally sized discs are assured by the characteristically normal embryonic development.

Types of atrophy

For purposes of discussion optic atrophy is usually divided into (1) primary atrophy, (2) secondary atrophy, and (3) atrophy following retrobulbar neuritis. The differential diagnosis between primary and secondary optic atrophy is not always clear-cut. Friedman[5] found that only one third of a series of normal optic discs possessed the classically normal appearance; two thirds showed characteristics which might under certain circumstances be interpreted as postinflammatory. In nearly 10 percent of his cases the appearance of the nervehead in the less typical eye was so uncharacteristic as to suggest, in the presence of optic atrophy, that there had been a neuritis. When all of the diagnostic points were taken into consideration, it could be assumed that there might have been difficulty in ruling out previous neuritis in two thirds of the eyes if optic atrophy had been present.

There has been a good deal of variation in the percentage of primary and secondary optic atrophy in the several series that have been reported. Masters[3] designated 24.6 percent of his series as primary and 75.4 percent as secondary, but in our University of California series there was a much higher percentage of primary disease.

The optic atrophies that follow retrobulbar neuritis, in which there has been no involvement of the disc itself, may appear to be primary in character, but since they have a fundamentally different underlying cause they are considered as a separate group of cases.

In addition to the specific conditions responsible for the three types of atrophy listed above, there are a number of rare heredofamilial diseases with which one or another of these types may occasionally be associated. In the following discussion these diseases will be considered under a separate heading.

Primary optic atrophy

Congenital hydrocephalus. Ford[9] suggests that congenital hydrocephalus should be mentioned first among the variety of conditions that may produce optic atrophy in children. The atrophy must be due to increased intracranial pressure but on many occasions papilledema does not develop and the disc then presents the picture of primary optic atrophy.

Basilar brain inflammations. Brennemann[10] states that the commonest cause of primary optic atrophy in children is the involvement of the intracranial portion of the optic nerve in such basilar brain inflammations as meningitis. The optic atrophy is really secondary to an optic neuritis but the nerve is involved so far from the nerve head that little or no evidence of the connection can be observed with the ophthalmoscope. Ford[9] states that in the chronic basilar type of brain inflammation optic atrophy develops in at least 51 percent of all cases and is usually primary.

Congenital neurosyphilis. Primary optic atrophy is common in congenital neurosyphilis. The onset may be at any age but is rare before two years. Woods[11] refers to it as the most important complication of the disease and states that it appears roughly from the onset of puberty to early adult life. He gives the general incidence of optic atrophy as about 15 percent and its incidence in congenital tabes as 50 percent. The peripheral field is affected first. Central vision is preserved until the last in most cases, but there may be a great variety of field defects, including central scotomas. The condition may progress very rapidly and blindness may ensue within a relatively few months after the onset of symptoms in spite of energetic treatment.

Syphilis does not occupy the prominent place among the causes of optic atrophy in infants and children that it does in adults. Green[12] found six cases of bilateral optic atrophy and two of unilateral optic atrophy in a group of 100 children with congenital syphilis. With better prenatal examination and care, and a reduction in the incidence of neurosyphilis as a result of earlier and more effective treatment of congenital syphilis, a decrease in the incidence of optic atrophy is to be expected.

This trend is borne out by data obtained from Miss Gertrude Karnan,[13] head teacher of the California State School for the Blind. Her figures are based only on the number of positive Wassermann reactions reported by the school hospital. There were probably other cases in which the cause of blindness was luetic in character but the disease had been arrested before the children reached the school so that the Wassermann reactions were negative. In the year 1930-31, in which there was an enrollment of 121, 7.4 percent of the cases of blindness were diagnosed as luetic on the basis of positive Wassermann reactions; in 1940-41, in which there was an enrollment of 139, this percentage had dropped to 2.8 percent; and in the year 1949-50 it had dropped to only 1.1 percent of the enrollment of 175 children.

Harrison[14] reported a 15-year-old colored girl suffering from congenital neurosyphilis who developed an osteoma involving the sphenoid bone. This effected complete closure of the optic foramen bilaterally with resultant primary optic atrophy.

Neoplasms. Primary optic atrophy may be produced by neoplasms which press directly upon the optic nerves at the chiasm. According to Ford[9] the commonest tumor of childhood in this region is the suprasellar cyst, which gives rise to more or less typical bitemporal hemianopia, although the field defects are not so constant or so typical as those associated with adenoma of the pituitary gland which is relatively rare in childhood. Such cysts may also cause papilledema without hemianopia, or may give rise to homonymous hemianopia.

Pressure on the optic nerve by either a suprasellar tumor or a subfrontal tumor may cause a central scotoma and produce the picture of retrobulbar neuritis. Malignant tumors of the pharynx, while rare, have been known to occur in children between the ages of six and 15 years.

Ford[9] states that in at least one third of these cases the nervous system becomes involved and that the neurologic symptoms are then frequently the first to be discovered. As a late sign the tumor may compress the optic nerve and cause atrophy.

Birth Injury. Optic atrophy may result from various types of birth injury. According to Juler,[15] the atrophy from such causes is usually unilateral, but at least one bilateral

case has been observed by Vannas.[16] According to Walsh,[17] Holt,[18] and others, birth injuries which may give rise to optic atrophy include (1) hemorrhage into or within the optic sheaths; (2) hemorrhage into the orbit in the region of the optic foramen, or more posteriorly as a result of fracture of the base of the skull; (3) dislocation of the eye; (4) avulsion of the optic nerve; (5) fracture of the orbit involving the nerve; and (6) exophthalmos following orbital bleeding.

Postnatal injury. Accidents producing such injuries as (1) fracture of the skull with laceration of the optic nerve, (2) compression following hemorrhage into the optic nerve sheath, and (3) avulsion of the optic nerve, may all lead to optic atrophy and loss of vision.

Diabetes. Wolfram[19] reported optic atrophy in diabetes in four siblings whose ages ranged from 18 months to seven years. In discussing Wolfram's report, Wagener suggested that the gradual onset of loss of vision and its steady progression did not justify a diagnosis of Leber's disease. In his opinion the lesion was a bilateral, progressive, primary optic atrophy.

Tyrer[20] reported a boy, aged 18 years, with infantilism, goiter, diabetes mellitus, mental deficiency, and bilateral primary optic atrophy. The diabetes was discovered at eight years of age. A sister, aged 10 years, had primary optic atrophy associated with diabetes which had first been recognized at the age of three years. The parents were first cousins.

Lead poisoning. Primary optic atrophy may be caused by lead poisoning. Children are particularly susceptible. In the 40 cases seen by Ford,[9] the poisoning was the result of ingestion of lead paint gnawed from toys and cribs. Fukushima and Matsumoto[21] listed as sources the playing with toys painted with lead paint and the biting of fingernails by children living in painted houses. Lead is also present in some face powders, which may be a source of infant poisoning if used by nursing mothers. Papilledema and optic neuritis may occur in these cases, sometimes simultaneously (Walsh[17]). The prognosis is not good and when there is a secondary atrophy there is likely to be severe and permanent loss of vision.

Drug poisoning. A number of toxic substances have been incriminated as causes of optic atrophy.

Aspidium, a toxic drug used in the treatment of tapeworm, is usually not absorbed by the digestive tract, but when it is, the toxic effects are likely to be very pronounced. If sufficiently large doses are taken there may be a convulsant action on the spinal cord; Sysi[22] reported a case of resultant optic atrophy.

Wood alcohol and arsenic have been designated as very exceptional causes of primary optic atrophy in childhood.

Tryparsamide has occasionally caused severe damage to the optic nerve. The impossibility of checking visual fields properly led Walsh[17] to the conclusion that tryparsamide should not be used in children. Thallium, which is used for epilation in the treatment of fungus infections of the scalp and as a depilatory cream, may cause optic atrophy,[23] Ford[9] says that thallium induces a selective atrophy as a rule and that in the cases seen by him it has given rise to central scotomas with temporal atrophy.

Macrocephaly. This is an extremely rare condition in which hypertrophy of the brain and a symmetrical enlargement of the skull are associated with varying degrees of mental deficiency. As Walsh[17] points out, macrocephaly is a morbid state not to be confused with physiologically large skulls which may be associated with unusually high intellectual attainments. The condition is not related to hydrocephalus and the occasional occurrence of two cases in one family suggests that there may be a familial tendency. Ford found optic atrophy in three siblings with this disease.

Osteopetrosis (Albers-Schonberg disease, marble bones). This condition is characterized as follows: (1) By increased density and thickness of both the cortical and spongy bones; (2) by spontaneous fracture; and (3) at times by changes in the blood resembling

those seen in anemia or leukemia. Brennemann[10] states that osteopetrosis will cause primary optic atrophy by direct pressure on the optic nerve.

Riser[23] noted that if patients with the disease were 15 years of age or older the eyes were not involved, but that if they were younger than 10 years at the time of onset, a large percentage developed primary optic atrophy.

Other cases of optic atrophy associated with this condition have been reported by Anthony and Pollack[24] and others. Papilledema was reported in one case.

Dwarfism associated with prognathism, thickening of the skull bones, skeletal changes, peculiar retinal pigmentation, deafness, mental deficiency, blue extremities, and optic atrophy was reported by Cockayne.[25] The condition was found in two siblings whose fundi showed bilateral primary optic atrophy with small retinal vessels.

Secondary optic atrophy

The term "secondary optic atrophy" refers to the atrophy that is associated with changes on the disc that follow either a papilledema or an optic neuritis.

Atrophy secondary to papilledema

Congenital hydrocephalus. As has already been stated, hydrocephalus often causes a primary optic atrophy. In many instances, however, it produces a papilledema followed by secondary atrophy.

Neoplasms. Intracranial neoplasms are among the commonest causes of increased intracranial pressure in childhood. As a rule the pressure is due to obstruction of the ventricular system so that the location of the tumor determines whether signs of increased pressure develop early or late (Ford[9]). In addition to neoplasms, other space-occupying masses, such as cysts, granulomas, abscesses, and hematomas, may cause increased intracranial pressure with resultant optic atrophy.

Lead poisoning. According to Ford,[9] obstruction of the venous circulation by lead poisoning can produce a papilledema followed by optic atrophy.

Insect bite. Strong[26] reported a case of bilateral secondary optic atrophy in a 15-year-old boy after an insect bite on the left side of the face. The bite was followed by swelling, painful headache, a fever of 105°F., and unconsciousness. The retina was edematous and there were many hemorrhages and papilledema.

Craniostenosis. The craniostenoses form a group of conditions in which cranial deformities have resulted from abnormal fusion of bones of the skull. In several types—oxycephaly, scaphocephaly, and Crouzon's disease—optic atrophy has been reported, as follows:

Oxycephaly (tower skull) is a premature synostosis of the cranial sutures resulting in a deformed head, increased intracranial pressure, and visual loss from optic atrophy. Greig[27] divided oxycephaly into three groups: (a) true oxycephaly recognizable at birth and due to generalized craniofacial synostosis; (b) delayed oxycephaly due to premature union of the cranial bones during infancy; and (c) false oxycephaly, a localized process due to localized disease of the cranial bones. Loss of vision from optic atrophy is a frequent complication.

Walsh[17] feels that the increased intracranial pressure from the oxycephaly results in a secondary optic atrophy and in a mechanical stretching of the optic nerves. Lisman[28] also regards the atrophy as usually due to traction on the optic nerve and rarely to stenosis of the optic foramen. According to Lisman, oxycephaly is differentiable from craniofacial dysostosis by virtue of (a) the absence of an hereditary factor in oxycephaly, (b) its high incidence among males, and (c) its diffuse nature. In oxycephaly, moreover, there is no depression of the sella.

Scaphocephaly is a condition in which the skull is characterized by a projecting, keel-like, sagittal suture resulting from premature ossification. This premature closure of the sagittal suture allows for further expansion in the anterior-posterior and vertical

diameters only. According to Walsh,[17] optic atrophy is a common complication.

Craniofacial dysostosis (Crouzon's disease) is a premature synostosis of the sutures of the base of the skull. The picture is characterized by a central prominence of the frontal region including a peculiar nose resembling a beak. Prognathism, exophthalmos, and external strabismus are also present, and there may be papilledema followed by optic atrophy. There are various theories as to the etiology of the atrophy. Vorisek,[29] among others, considers it a result of intracranial pressure; another group, to which Krause and Buchanan,[30] belong, regard it as a result of mechanical traction on the nerves due to the length of the canals; and Behr's opinion[31] is that the shift of the optic canal back over its floor produces pressure on the nerve by the carotid artery. Parks and Costenbader[32] reported two cases in which the intracranial pressure was relieved by surgery with consequent elimination of the papilledema.

Encephalitis periaxialis diffusa (Schilder's disease). This disease affects the white matter of the cerebral hemisphere in such a way that there is widespread demyelinization followed by destruction of the axis cylinders. The greatest change usually takes place in the white matter of the occipital lobes. A progressive spastic paralysis, blindness, and death ensue. According to Rea,[33] mild papilledema occurs in 25 percent of cases. In rare instances there may be a primary optic atrophy. Berliner[34] found that in 90 percent of the cases with amaurosis, bilateral optic atrophy was observed before death.

Exophthalmos. Optic atrophy may follow orbital bleeding in the so-called "exophthalmos of the newborn."

Atrophy secondary to optic neuritis

Secondary optic atrophy may follow any severe optic neuritis. The neuritis may be strictly a papillitis, in which the process actually involves the optic disc, or it may be the type in which the reaction is behind the lamina cribrosa but extends far enough forward to cause edema of the disc. It may have the appearance of a papilledema but be distinguishable from it by the other signs of optic neuritis; that is, central scotoma and rapid loss of vision. Optic neuritis in association with a number of the diseases of childhood may give rise to optic atrophy, as follows:

Meningococcus meningitis, as Dunphy[35] points out, is not infrequently complicated by an optic neuritis. In most cases the vision returns to normal as the infection subsides, but the disc may remain pale and the vision be much better than the appearance of the disc would suggest. Nettleship[36] noted that complete blindness is an early manifestation of meningococcus meningitis in infants. Although most patients recover, there is an occasional optic atrophy.

Measles may produce the same picture. Meurer,[37] for example, reported three boys with encephalomyelitis following measles in whom there was a bilateral optic neuritis followed by changes that suggested secondary optic atrophy, although vision ranged between 6/9 and 6/6.

Chickenpox followed by encephalomyelitis may produce optic neuritis, which may be followed in turn by optic atrophy with varying degrees of blindness. There is a high degree of recovery from the optic neuritis, but even though the vision may be normal, or nearly so, the disc may be white. Two such cases were reported by Walsh.[17]

Smallpox has been reported as producing a similar picture.[17]

Mumps followed by encephalomyelitis may affect the optic nerve variously. Optic neuritis, neuroretinitis, retrobulbar neuritis, papilledema, and optic atrophy have all been reported. As Ford[9] pointed out, some of these conditions may be due to local inflammatory processes but there is no doubt that others are a result of increased intracranial pressure.

Optic atrophy following retrobulbar neuritis

This type of atrophy, in which there has been no involvement of the disc itself, may

occur in measles, vaccinia, and other conditions which may be associated with acute disseminated encephalomyelitis. Other localizations of infection may produce a similar picture. I have seen one child in whom an acute infection of the ethmoids caused an atrophy, and Ford[38] reported optic atrophy due to paranasal sinusitis after scarlet fever.

Avitaminosis. In a study of defective vision in schoolchildren in Nigeria, Moore[39] reported retrobulbar neuritis followed by both partial and complete optic atrophy as a result of avitaminosis. The affected children presented a syndrome of sore tongue and mouth, scrotal or vulval rash, and optic atrophy.

Disseminated sclerosis is characterized by disseminated areas of myelin destruction. In adults there are three common forms of onset: (1) With cerebral symptoms; (2) with paraplegia; and (3) with retrobulbar neuritis. Ford[9] has seen children between the ages of nine and 15 years typically affected with this disease but with the onset limited exclusively to retrobulbar neuritis. Complete optic atrophy with blindness has been known to occur but is uncommon.

Encephalitis periaxialis diffusa (Schilder's disease). As already described in the section on optic atrophy secondary to papilledema, Schilder's disease is a demyelinization accounting for massive lesions in the subcortical white matter of the cerebral hemispheres and producing a progressive spastic paralysis, blindness, and death (Berliner[24]). The blindness is cerebral in type, due to bilateral involvement of the occipital lobes.

Krabbe's acute infantile diffuse cerebral sclerosis. As has been pointed out by Walsh,[17] there are several disease processes in infants that are characterized by massive demyelinization of the white matter of the brain and by secondary gliosis. As a group these cases are designated as diffuse or centrolobar sclerosis and are undoubtedly related to Schilder's disease. In one form of this condition, known as Krabbe's acute infantile form,[40] universal rigidity of the musculature and violent tonic spasms develop at about the fifth month; later there is extensive paresis and pronounced disability, and later still optic atrophy.

Intracranial tumor. An intracranial tumor pressing directly on the intracranial portion of the nerve, which may give rise to scotomas for a long time before more extensive defects in the fields appear, must be considered a possible cause of optic atrophy.

Heredofamilial diseases which produce optic atrophy

There are a number of relatively rare heredofamilial diseases occurring in infancy, childhood, and adolescence which may give rise to one or another of the various types of optic atrophy already discussed.

Leber's disease was first described by Leber[41] in 1871. He defined it as an hereditary amaurosis resulting from neuritis of the optic stem which in the beginning appeared either as a retrobulbar neuritis or as a neuroretinitis. In either event the neuritis progresses to partial optic atrophy, and occasionally to complete optic atrophy.

While the disease attacks primarily the male members of the family, Cordes[42] found from a review of the literature that approximately 20 percent of the cases occur in females. The condition usually develops between the 18th and 25th years but has been reported a good many times at an earlier age; Ford[9] found one case in a child five years of age.

In the early stages the fundus may be normal or may show a mild optic neuritis with a partial veiling of the vessels. Characteristically the field presents a central scotoma without peripheral contraction. In the later stages the optic nerve shows temporal atrophy that occasionally progresses to complete atrophy. Scott[43] found 61 cases of Leber's disease with optic atrophy in three families totaling 250 members. Bell[44] found historical evidence of the disease through seven, eight, and even 11 generations.

Congenital optic atrophy is now generally accepted as a term referring to the complicated heredofamilial bilateral atrophy de-

scribed by Behr[45] in 1909. The degree of severity varies from complete atrophy to involvement of the papillomacular bundle only. In the cases described by Behr the atrophy was associated with mild disturbances of the pyramidal tracts resulting in hypertonicity, increased reflexes, ataxia, bladder disturbance, and a degree of mental deficiency. Behr felt that the complications of the condition served to differentiate it from Leber's disease, which is an uncomplicated, juvenile familial optic atrophy. Walsh and Ford[46] reported congenital optic atrophy in two members of a family in whom it was limited to a pronounced atrophy of the papillomacular bundle.

Not all cases are clear-cut. For example, Griscom[47] described a family which he believed to be afflicted with Leber's disease; Walsh[17] reviewed the same cases and felt that they were probably congenital optic atrophy. As Walsh points out, it is clear that the diagnosis of congenital optic atrophy can rarely be made with certainty and must depend largely upon the history and pedigree. Both eyes are always affected. In any unilateral case it would seem impossible to eliminate birth injury as a factor.

The importance of the family history has been stressed by Franceschetti[48] who pointed out that although the disease may appear in several members of one family, it has not been observed in several generations of the same family.

Dysostosis multiplex (Hurler's disease). In a review of the literature, Cordes and Hogan[49] found only two cases of optic atrophy as a complication of Hurler's disease. These cases were described by Davis and Currier[50] and in each instance there was bilateral complete optic atrophy. A complicating factor, however, was the association of the condition with buphthalmos and hydrocephalus.

Chrondrectopia (Ollier's disease) is characterized by the presence of normal cartilage in abnormal places. Islands of cartilage which have escaped ossification may occur in certain bones of the skull, including the sphenoid. Neilson[51] reported a 17-year-old girl in whom the optic foramen, superior orbital fissure, and right sphenoid bone were involved, and whose left eye showed optic atrophy.

Congenital spastic diplegia (Little's disease) is a condition characterized by bilateral paralysis and spasticity from birth. Mental defects, involuntary movements, and ataxia are present in varying degrees and Walsh[17] has observed optic atrophy.

Athetosis. Walsh[17] has also reported an associated, nonprogressive, bilateral optic atrophy in a case of athetosis. This is a condition in which there are constantly recurring movements of the hands and feet and often of the body and head.

Friedreich's ataxia is an heredofamilial degenerative disease in which there is progressive degeneration of the spinocerebellar and corticospinal tracts, posterior columns, and posterior roots; optic atrophy occurs in rare instances only. Franceschetti[52] suggests that when it does occur it signifies the presence of two separate entities. In the hereditary cerebellar ataxia of the types of Sanger Brown and of Marie, the onset is between the ages of 16 and 35 years and early optic atrophy is the rule.[17]

Familial cerebral calcification. A family of four children with familial cerebral calcification associated with epilepsy was reported in 1929 by Geyelin and Penfield;[53] one of the children had a primary optic atrophy.

Amaurotic family idiocy. While the usual ocular changes in this disease are macular, optic nerve atrophy was more pronounced than macular change in a case reported by Higier,[17] and Vogt[17] reported three cases of the late infantile form that displayed optic atrophy in addition to the usual fundus changes. In their study of the ocular pathology of juvenile amaurotic idiocy, Givner and Roizin[54] described the findings in a 17-year-old-girl; in addition to the usual changes in the macular area, there was bilateral optic atrophy.

Hereditary spastic paraplegia. According to Rhein,[55] this heredofamilial degenerative

process of the pyramidal tracts, with progressive paraplegia, rather frequently produces optic atrophy.

The Laurence-Moon-Biedl-Bardet syndrome consists of pigmentary degeneration, which is usually typical, associated with mental retardation, obesity, and hypogenitalism, and in some instances with polydactyly. According to Walsh,[17] although the typical ocular changes are pigmentary, pronounced optic atrophy can occur, and when it does there is usually extensive atrophy of the choroid so that there appears to be a choroideremia.

Schüller-Christian-Hand disease is a diffuse liporeticular disease of young children characterized by degeneration of and lipoid deposits in the bones, especially those of the skull, and by exophthalmos and diabetes insipidus. Optic atrophy may occur as a result of pressure on the nerve, either in the orbit or within the cranium. It has also been reported as a sequela of papilledema.[17]

Recklinghausen's disease (neurofibromatosis), a disease with a hereditary tendency, manifests itself as diffuse proliferation of Schwann cells of the peripheral nerves. Clinically there is multiple neurofibromatosis widely disseminated throughout the peripheral nervous system with gliomatosis of the central nervous system associated with café-au-lait spots over the body. The lesion is noted first in childhood. Reese[56] states that there is no part of the eye and its adnexa which may not be involved.

Emanuel,[57] Goldman,[58] and others have pointed out the high percentage of these cases that have associated tumors of the optic nerve. It was the monumental work of Davis,[59] however, that clarified the picture. He has pointed out that these optic nerve tumors associated with Recklinghausen's disease are not neurofibromas but gliomas. They may involve any portion of the nerve and produce atrophy of the nerve.

According to Reese[56] 60 to 70 percent of all tumors of the optic nerve occur in children, sometimes at an early age. Davis[60] has seen an optic atrophy in a six months' old infant due to glioma in Recklinghausen's disease. Often the evidence of the associated disease consists only of café-au-lait pigmented spots or easily overlooked tumors of the skin. Davis[59] has emphasized the fact that unexplained blindness or primary optic atrophy not infrequently is due to early tumor formation of the chiasm or optic nerve. Careful search will usually reveal café-au-lait spots or easily overlooked tumors of the skin. Because the neurofibromas of the skin often do not develop until puberty the café-au-lait spots may be the only evidence of Recklinghausen's disease in these cases.

A survey of 81 cases of optic atrophy in children

A total of 102 cases of optic atrophy in infancy, childhood, and adolescence were observed at the University of California Hospital in San Francisco between 1920 and 1950. The histories of 81 of these cases contained sufficient data for the purpose of this study. The age range of the patients was from six months to 16 years.

Primary optic atrophy

The etiology of the primary optic atrophy which occurred in 43 of the 81 cases was as follows:

Congenital syphilis (eight cases). The eight patients with congenital syphilis ranged in age from three to 13 years; four were males and four were females. Both eyes were involved in each instance. The three-year-old child had a right-sided hemiplegia. It is noteworthy that only one new case of optic atrophy in congenital syphilis has been seen in this hospital since 1945.

Trauma (six cases). Trauma accounted for six cases of atrophy, all of them monocular. The ages at which the trauma occurred ranged from two to 16 years. The injuries included a basal fracture occurring in an automobile accident, a fracture of the right occipital bone, a severe blow to the eye with-

out fracture, and a penetrating injury. In two cases the injury was not described.

Congenital optic atrophy (five cases). The five patients with congenital optic atrophy ranged in age from six months to 10 years. The patients were all males and both eyes were always involved. Two were unquestionably of the type described by Behr but there was considerable doubt as to the proper classification of the other three.

Cerebral aplasia (three cases). In three instances the diagnosis was cerebral aplasia. All three showed mental retardation, all were males, and all had bilateral ocular involvement. Their ages ranged from six months to five years.

Neoplasms (four cases). Tumors accounted for four instances of atrophy. The ages of the patients ranged from four to 13 years. The atrophy was bilateral in all but one case in which the tumor was an astrocytoma of the right optic nerve. Two of the neoplasms were pituitary and one was a Rathke pouch tumor.

Craniopharyngioma (one case). A right-sided primary optic atrophy was observed in a 17-year-old boy in whom a diagnosis of probable craniopharyngioma was made but never confirmed.

Birth injury (one case). There was one case of a nine-year-old boy with bilateral optic atrophy, much more marked in the right eye, in whom there was a history of birth injury. There was mental retardation and the visual loss had apparently been aggravated by a fall on the head at three and one-half years of age. Inability to use the right hand followed this fall.

Congenital osseous dyscrasia (Morquio's disease; one case). A 13-year-old boy with Morquio's disease had a left-sided primary optic atrophy. The mother stated that two siblings were similarly afflicted.

Osteomyelitis (one case). A 17-year-old boy had a right primary optic atrophy which followed an osteomyelitis of the right frontal maxillary and mandibular bones. Culture showed hemolytic staphylococci.

Hemorrhage (one case). A one and one-half-year-old girl developed a bilateral primary optic atrophy after an attack of jaundice and purpura. Hemorrhage was believed to be the etiologic factor.

Toxoplasmosis? (one case). A four-year-old boy was seen in 1931 with a partial primary optic atrophy in the right eye and complete atrophy in the left. There was a history of convulsions from birth associated with mental retardation. The eyes also showed a bilateral central chorioretinitis with changes "suggesting some prenatal retinitis proliferans." These findings are strongly suggestive of toxoplasmosis, a disease not recognized at that time.

Cerebral spastic palsy (one case). A five-year-old girl had a cerebral spastic palsy associated with bilateral primary optic atrophy involving two thirds of the disc.

Unknown etiology (10 cases). There were 10 cases of bilateral primary optic atrophy in children ranging from six months to seven years of age for which no causal factor could be established. Four showed mental deficiency and two were classed as idiots.

As has already been stated, congenital hydrocephalus may on occasion present a picture of primary optic atrophy. Since the atrophy must be a result of increased intracranial pressure, however, and must therefore be considered secondary in nature, the cases in this series have been classified as secondary even though two of them could have been considered primary on the basis of clinical appearance.

Secondary optic atrophy

Congenital hydrocephalus (11 cases). Hydrocephalus was the largest single factor in the development of secondary optic atrophy in this series. The ages ranged from six months to 10 years and there were six cases in males and five in females. In five instances the condition was an internal hydrocephalus. The atrophy was bilateral in all cases and was secondary in type except in two patients in whom the fundus picture was that

of a primary optic atrophy. As noted above, this occurs not infrequently.

Neoplasms (five cases). The ages of the five children with intracranial tumors ranged from one and one-half to 15 years. There were three females and two males, and there was bilateral secondary optic atrophy in all five cases. In one instance the type of tumor was undetermined; the others included a gliomatous cyst in the cerebellum, a cerebellar tumor of the left lobe associated with an internal hydrocephalus, a pituitary tumor, and a posterior fossa tumor.

Subdural hematoma (one case). One patient, a one and one-half-year-old boy, with the symptoms of an intracranial tumor and a bilateral secondary optic atrophy, was found to have a subdural hematoma.

Encephalomyelitis (three cases). There were three cases in which encephalomyelitis caused a bilateral secondary optic atrophy. The youngest patient was six months old and the oldest 15 years. All had neurologic signs. The etiology was mumps in one instance, meningococcic meningitis in another, and tuberculous meningitis in the third.

Oxycephaly (three cases). The three cases of oxycephaly occurring in this series were six months, one year, and seven years of age respectively. The atrophy was bilateral in two cases and unilateral in the third.

Scaphocephaly (one case). There was one patient, a seven-month-old boy, who had scaphocephaly with bilateral optic atrophy associated with mental retardation and general aplasia.

Congenital lues (one case). A 13-year-old patient with congenital lues had a bilateral, postneuritic atrophy.

Cerebellar ataxia, Marie's type (one case). A seven-year-old boy who had never walked had a cerebellar ataxia of the type of Marie, associated with secondary optic atrophy in the right eye; the left eye was normal. The patient had been delivered with forceps. There was no heredofamilial history.

Petrositis (one case). A seven and one-half-year-old male had a secondary optic atrophy on the left side, associated with loss of hearing and running of the left ear. The disorder was diagnosed as petrositis.

Intracranial hemorrhage at birth (one case). An 18-month-old male had a secondary optic atrophy following a severe intracranial hemorrhage at birth.

Unknown etiology (four cases). There were four patients with bilateral secondary optic atrophy in whom the etiology was not established. In one, a 15-year-old female, a diagnosis of atrophy of the brain had been made. In another, an eight-year-old male, the atrophy was accompanied by a bilateral macular chorioretinitis, the description of which suggested toxoplasmosis.

Optic atrophy following retrobulbar neuritis

Leber's disease (four cases). In these four cases, Leber's disease had been transmitted from the maternal grandmother to four siblings. The 16-year-old male had a bilateral optic atrophy in which the temporal halves of the discs were most involved; the 11-year-old and 12-year-old (both males) showed definite bilateral temporal atrophy; and the nine-year-old female showed the earliest changes of temporal atrophy with the usual central field changes.

Meningococcus meningitis (one case). A 14-year-old female showed primary optic atrophy with complete blindness; she had had meningococcus meningitis at one year of age.

Lead poisoning (one case). A seven-year-old male developed bilateral retrobulbar neuritis, followed by bilateral optic atrophy, from lead poisoning.

Comments and Conclusions

1. The histories of 81 cases of optic atrophy in infancy, childhood, and adolescence observed at the University of California Hospital in San Francisco between 1920 and 1950 were analyzed for etiologic factors.

2. Of the 81 cases, 43, or approximately 53 percent, had primary optic atrophy; 32, or 39 percent, had secondary optic atrophy; and six, or eight percent, were the result

of retrobulbar neuritis. The percentage of cases of primary atrophy was much higher than it was in the blind school series reported by Masters, probably because many of the cases cited as primary in the Univerity of California series would not have reached a blind school because of early death.

3. The largest single cause of optic atrophy of all types was hydrocephalus (11 cases), with neoplastic disease second in frequency (10 cases). It is noteworthy that congenital syphilis was the third most frequent cause.

4. Cases of congenital optic atrophy and atrophy in association with hydrocephalus, cerebral aplasia, and oxycephaly appeared at an early age.

5. Cases of atrophy due to central nervous system lues, neoplasms, and trauma were found in the age group of nine to 11 years.

6. One case of meningococcus meningitis in a one-year-old infant bore out Nettleship's observation that complete blindness is an early manifestation of this disease when it occurs in infants.

7. Of special interest was the report from the California State School for the Blind which showed that the percentage of blind with congenital syphilis had dropped from 7.4 percent in 1930 to 1.1 percent in 1950. In 1914 Bishop Harman stated that 56 percent of the cases of optic atrophy in the London School for the Blind had congenital syphilis. Only one new case of atrophy in association with congenital central nervous system syphilis has been seen at the University of California since 1945. In December, 1950, a 13-year-old Negro boy, newly arrived from the "deep south," reported to the clinic with primary optic atrophy.

8. Exclusive of the cases of traumatic origin, 35 percent of all cases of primary optic atrophy showed mental deficiency; this associated feature was not recorded in the patients with secondary optic atrophy.

9. It was clear from this study, which was based on a review of the literature and an analysis of 81 cases, that optic atrophy in infancy, childhood, and adolescence may arise from an exceedingly wide variety of causes.

384 Post Street (8).

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CATARACT EXTRACTION IN THE ONE-EYED PATIENT*

A STUDY OF 103 CONSECUTIVE CASES

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The purpose of this study was to determine the effectiveness of the round-pupil intracapsular cataract extraction in a series of patients, each of whom had developed a mature cataract in his only eye. It was thought that careful analysis of the significant details of each case, both successful and unsuccessful, might lead to an increase in the incidence of successful cataract extractions.

For material, I used 103 consecutive one-eyed patients in a larger series of 522 cataract extractions performed by me between January, 1947, and July, 1951. The term "one-eyed" refers to the patient who has had one eye removed, or if retained, the vision in it has been reduced to less than 10/400 with a nonremedial defect.

The various psychologic aspects of such patients who must have their lens removed have been thoroughly presented by Kirby[1] and are not considered herein.

If the nonseeing globe has been retained, it is carefully studied for complications which might affect the outcome of surgery on the better eye. If it is glaucomatous, the examiner must determine whether it is of the primary type or secondary to causes such as cataract extraction or chronic uveitis. Dystrophy of the cornea in the nonseeing globe requires a meticulous study of the better eye to ascertain if it is similarly involved. If the eye has been removed, a searching history of the details of its loss must be obtained.

Tabulation of the causes of the loss of vision in the first eye shows that complications of cataract extraction are the largest single group. Primary glaucoma and physical trauma are next in frequency (table 1).

The causes of the loss of the first eye from complications after catract extraction were studied in meticulous detail before the second eye was operated on (table 1a). I was able to prevent the same complication that had occurred in the first eye from developing in the second eye in all types except one retinal detachment and three of the corneal dystrophies.

TABLE 1

CAUSE OF LOSS OF VISION OF FIRST EYE IN 103 PATIENTS

Complications following cataract extraction (see table 1a)	36
Primary glaucoma	26
Physical trauma	23
Macular degeneration	3
Chorioretinitis, central	2
Intraocular hemorrhage	2
Retinal detachment, idiopathic	1
Amblyopia ex anopsia, severe	1
Optic atrophy after meningitis	1
Optic atrophy, cause unknown	1
Diabetic retinopathy	1
Endophthalmitis, cause unknown	1
Corneal degeneration from repeated ulcers	1
Cause unknown	4

TABLE 1a

LOSS OF FIRST EYE FROM CATARACT EXTRACTION COMPLICATIONS (36 CASES)

		Number with Same Complication in Second Eye
Cataract extraction followed by:		
Operative infection	9	0
Secondary glaucoma, uncontrollable (from causes such as incarceration of iris or incomplete removal of cortex)	7	0
Retinal detachment	4	1
Phthisis bulbi (vitreous loss with inflammatory reaction)	3	0
Severe uveitis	3	0
Intraocular hemorrhage	3	0
Hemorrhagic glaucoma and corneal dystrophy	2	1
Corneal dystrophy	4	3
Epithelial downgrowth	1	0

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Loss of first eye from cataract extraction

In 31 of the 36 cases in which the first eye was lost after a cataract extraction, the surgery was performed elsewhere.

Four were patients upon whom I had performed the cataract extraction in the first eye in my total series of 522 cataract extractions. Two of these were cases of operative infections and it was necessary for me to eviscerate the eyes. My gratification in removing the cataract from the second eye of each of these patients with the return of 20/20 vision to each is understandable. In the third eye, vitreous was lost at surgery, and this was followed by secondary glaucoma, finally corneal dystrophy. When this patient's other lens became cataractous, I extracted it without complication, and 20/20 vision was obtained.

My fourth loss occurred in a patient with uncontrollable glaucoma secondary to the cataract extraction with incarceration of the iris pillars in the wound and severe uveitis. At the cataract extraction upon her second eye, fluid vitreous filled the anterior chamber immediately upon the completion of an uncomplicated corneal section. However, the cataract was removed intracapsularly, a peripheral iridotomy was performed, visible vitreous loss did not occur, no iris incarceration developed, and the patient's corrected vision three years after surgery is 20/40.

A fifth patient of special interest to me was one whose lenses, hitherto clear, developed cataracts within four months after a corneoscleral trephination for glaucoma was performed on both eyes by a member of our resident staff. One eye was lost but corrected vision of 20/20 with full field was retained by the other eye and the tension was finally controlled, although three antiglaucoma operations were required.

Glaucoma complicating cataract

As tabulated in Table 1, the vision of the first eye had been lost from primary glaucoma in 26 patients. In all of these, the second eye was similarly involved in varying degrees.

TABLE 2

Visual results

20/20	25		
20/25	21	=75%	
20/30	18		
20/40	13		
20/50	3		
20/60	2		
20/70	2	=12%	
20/80	2		
20/300	3		Considered individually in Table 2a
20/300	1		
7/200	1		
4/200	1		
F.C. 5′	1		
F.C. 3′	1	=13%	
H.M. 1′	2		
Light projection or perception only	4		
No light perception or projection	3		

In this total of 26 eyes involved both by glaucoma and cataractous lens changes, 13 obtained visual acuity of 20/40 or better, and 13 obtained vision of less than 20/40. In the latter group, most had extremely poor vision. Pertinent details of these cases are tabulated in Section I of Table 2a. It must be remembered that the surgeon begins with an eye damaged by glaucoma, and that this is obscured at first by the cataractous lens.

A study of lateroinferior cataract extraction after antiglaucoma surgery has been recently reported.[2] A study of simultaneous antiglaucoma surgery and cataract extraction versus a two-stage procedure of antiglaucoma surgery followed after six months or more by extraction of the cataractous lens is planned.

Retinal detachment after cataract surgery

Evidence that retinal detachment is more likely to follow extracapsular extraction or intracapsular extraction is insufficient in this small series to permit any conclusion. However, I have developed a surgical procedure to attempt to prevent retinal detachment ocurring in the second eye, as follows:

In eyes that have developed retinal detachment after cataract extraction in the first eye,

and in which peripheral cystic degeneration has been or can be visualized in the second eye, I apply surface diathermy to the sclera over the ora serrata completely encircling the eye. After two months or more have elapsed, the cataractous lens is removed from the eye. In two such eyes, now followed for two years, detachment has not occurred.

Corneal dystrophy

There is neither effective prevention against corneal dystrophy developing after cataract extraction nor adequate treatment if it does develop. In four patients, the loss of vision in the first eye was caused by corneal dystrophy which followed cataract extraction; in three of these, corneal dystrophy advanced in the second eye operated upon for cataract to such a degree that useful vision was lost.

If a cataract develops in an eye which already has required antiglaucoma surgery and corneal dystrophy has developed, the extraction of the lens may upset the control of the glaucoma, and is likely to stimulate further advance of dystrophy.[3] Corneal dystrophy contraindicates cataract surgery unless the vision is reduced to light projection or the lens becomes intumescent and causes secondary glaucoma. In such cases, when corneal dystrophy is present, it is better to section the cornea slightly scleralward above the upper limbus. Although the section goes through the filtering operation, it interferes with the corneal metabolism as little as possible, since it leaves undisturbed the lower limbus through which the cornea is receiving nutrition.

Unpredictability of visual acuity attainable

In patients with hypertension, diabetes, and macular degeneration, it is impossible to predict the amount of vision that will be obtained in an eye in which the lens is completely cataractous. Though the results are generally poor, surgical extraction should not be withheld if there is any chance of improvement.

A 73-year-old patient who had sustained a cerebral hemorrhage from severe hypertension showed multiple retinal hemorrhages in the right eye with only light perception for six years. The lens in the left eye had been completely cataractous for two years, but light projection was normal. Corrected vision of 20/30 has been retained by the patient for two and one-half years since the cataract was extracted.

Another patient obtained and has retained for three years vision of 20/30 after cataract extraction in the second eye, though the first was reduced to 20/400 from senile macular degeneration.

A 63-year-old diabetic, requiring insulin with restricted diet, had, four years previous to my first examination, an extraction of the cataractous lens from the right eye, followed by hemorrhage, atrophy of the eye, and eventually enucleation. After the vision had been reduced to light projection, the cataract was removed and the patient has retained corrected vision of 20/40 for three years.

Vitreous hemorrhage occurring postoperatively gives a poor but not necessarily negative prognosis for vision. One patient developed a vitreous hemorrhage in the first few days after operation and the vision was reduced to poor light projection. Routine postoperative management was given, except that potassium iodide was prescribed orally. Whether this was of value or not is unknown but after six months the corrected vision was 20/25, and no other treatment was rendered.

Results

Good visual results in a cataract extraction series which includes multiple diseases in some of the eyes are understandably less than those in an uncomplicated series (table 2).

Visual acuity of 20/40 or better was obtained in 75 percent. A study of the 26 eyes obtaining less than 20/40 vision shows that nearly half of this group were involved with primary glaucoma, quite advanced before the cataract extraction (table 2a). Conditions existing prior to cataract extraction that could not be discovered until after surgery

TABLE 2a

A STUDY OF THE CASES IN WHICH VISION LESS THAN 20/40 WAS OBTAINED IN THE SECOND EYE AFTER CATARACT EXTRACTION (26 EYES)

Patient	First Eye	Second Eye	Preop. Vision	Postop. Vision
I. Primary Glaucoma, Chronic Noncongestive (13 eyes)				
(a) With antiglaucoma surgery prior to cataract extraction				
				(2–4 year follow-up)
1. M. C.	Enuc., abs. glau.	C.S. trephin. 6 yr. prior to cat. ext.	L.P.	20/300
2. L. C.	Enuc., following physical trauma	Ant. sclerectomy 2 yr. prior c.s. trephin. 6 mo. prior to cat. ext.	L.P.	L.P., advanced optic atrophy discovered
3. L. D.	Enuc., abs. glau.	C.S. trephin. 2 yr. prior to cat. ext.	10/400	20/60
4. S. J.	Blind, abs. glau.	C.S. trephin. 2 yr. prior to cat. ext.	L.P.	H.M. 1′
5. J. F.	Enuc., abs. glau.	C.S. trephin. 3 yr. prior to cat. ext.	F.C. 3′	H.M. 1′
6. J. P. W.	Enuc., abs. glau.	C.S. trephin. 8 mo. prior to cat. ext.	20/100	20/80
7. B. Y.	Enuc., abs. glau.	5 oper. prior to cat. ext., tension never controlled	L. proj.	L. proj.
8. J. F. A.	Blind with glau. cat. and corneal dys.	Iridectomy 1 yr. prev.	L. proj.	L. proj.
(b) Antiglaucoma surgery performed simultaneously with extraction of cataractous lens				
9. L. B.	Enuc., abs. glau.	Iridectomy and Holth sclerectomy combined with cat. ext.	L. proj.	F.C. 5′
10. J. A. F.	Blind, abs. glau.	Trephin. and iridectomy combined with cat. ext. c.s. trephin. combined with cat. ext.	F.C. 4′	20/60
11. W. W. H.	Enuc., abs. glau.	C.S., trephin. combined with cat. ext.	L. proj.	20/50
12. C. E. H.	Enuc. following physical trauma	Iridectomy and sclerectomy combined with cat. ext.	20/80	20/80
13. N. J. M.	Blind, abs. glau.	C.S. trephin. and wide iridectomy combined with cat. ext.	20/80	20/50 but diacission was necessary
II. Corneal dystrophy (2 eyes)				
14. A. N.	Aphakia & corneal dys.	Corneal dystrophy	L. proj.	20/200 corneal dys.
15. W. T. W.	Severe trauma; corneal scarring & dys.	Corneal dystrophy	20/200	4/200 corneal dys.
III. Senile macular degeneration (2 eyes)				
16. E. P.	Cat. ex. followed by corneal dys.	Senile macular degen. undiscovered prior to surgery	L. P. & L. proj.	20/200
17. L. P.	Blind, abs. glau.	Senile macular degen. with temporal optic-nerve pallor	3/200	20/50
IV. Extreme myopia with extensive chorioretinal atrophy (1 eye)				
18. J. M.	Enuc. 13 years prior for retinal detach	Advanced myopia, with myopic degen., extracap. cat. ex. followed by severe iridocyclitis	12/200	No L. proj. (sec. glau followed by iridocyclitis and finally retinal detach.)
V. Old Hemorrhage of Retina from Vascular Disease Predating Surgery (2 eyes)				
19. F. P.	Pri. glau., advanced	Mult. macular hemor. from hypertension (2 stage procedure for c.s. trephin. and cat. ext.)	L. proj.	7/200
20. L. W.	Optic atrophy	Arteriosclerotic hemor. in macular area	H.H. 12″	20/70
VI. Technical Errors at Surgery (2 eyes)				
21. H. A.	Chronic glau. secondary to old iritis	C.S. trephin. & sclerectomy 9 yr. prior. At surgery, loss of vitreous bead	L. proj.	No L. P. Detachment of retina occurred after 3 mo.
22. H. G. W.	Chorioretinitis	Loss of vitreous, iris incarceration, chronic uveitis and corneal dys.	20/200	L. proj. corneal dys. occurred
VII. Postoperative Vitreous Hemorrhage (2 eyes)				
23. R. B.	Enuc., sec. glau. following cat. ex.	Vitreous hemor. occurring 24 hr. after surg.	L. proj., L.P., and H.M.	No L. P.
24. E. McR	Corneal dys. following cat. ex.	Vitreous hemor. 6 days postop. with secondary glau. and corneal dys.	H.M.	F.C. 3′
VIII. Miscellaneous (2 eyes)				
25. L. S.	Traumatic destruction	Optic nerve atrophy	L. proj.	20/70
26. M. M.	Infection following cat. ex. evisceration required	Cerebral atrophy	L. proj.	20/200

included senile macular degeneration and hypertensive macular hemorrhages. Causes that might have been avoided with different management included corneal dystrophy and postoperative vitreous hemorrhage.

TECHNIQUE

PREOPERATIVE PREPARATION

These patients are admitted to the hospital 48 hours prior to operation. Cultures are taken of the lid margins and conjunctivas

upon admission. The blood-agar and milk-agar plates of these cultures are read by a bacteriologist shortly before the operation is to be performed. Milk-agar plates are used to facilitate identification of Staphylococcus aureus. Colonies of Staphylococcus albus and diphtheroids are almost invariably found. If organisms usually considered to be pathogenic are found, the operation is delayed, the indicated treatment begun, and after a few days another culture is repeated. Not until a culture negative for these pathogens is obtained is surgery performed. In 13 of the cases, the operation was delayed. In four of these, the first eye had been lost by an infection following cataract extraction.

Certain bacteria as B. proteus are quite resistant to antibiotics and several weeks were required to eliminate them with aureomycin and streptomycin. However, treatment with Aerosporin for a few days is sufficient to eliminate B. proteus. Hemolytic Staphylococcus aureus of the lids requires treatment with aureomycin ointment for a short time only.

For cleansing of the lids, the cocoanut-oil derivatives are quite superior to tincture of green soap. Because of its bacteriostatic qualities, we scrub the patients' eyelids, 48 hours preoperatively, with pHisoderm or Formula 99, then 24 hours preoperatively, and then again immediately preceding surgery.

On the preoperative day, the lacrimal passageway is irrigated with pencillin (solution, 50,000 units per cc.). Prophylactic administrations of penicillin (400,000 units) and streptomycin (1.0 gm. in three doses daily) are given parentally on the day before, the day of, and the day after operation; these drugs are discontinued three days after surgery.

Aureomycin ointment ophthalmic is instilled into both eyes in the afternoon and the early evening before the operation. In the eye to be operated upon, homatropine (5.0 percent) in zephiran chloride aqueous (1:5,000) is instilled twice on the night before operation. A sterile dressing is applied over the eyelids of the eye upon which the operation is to be performed.

Sedation is obtained by giving pentobarbital sodium (1.5 gr.) the night before operation, and repeating it the next morning two hours before the operation. An hour before operation, demerol (50 mg.) is given intramuscularly.

On the morning of operation, homatropine (5.0 percent) is instilled into the eye to be operated upon. Thirty minutes before operation, several drops of neosynephrine hydrochloride (solution, 10 percent) are instilled. Tetracaine hydrochloride (0.5 percent) is instilled every few moments for four or five times before the incision is made.

The surgical field is scrubbed with pHisoderm or Formula 99, irrigated with sterile normal saline, and dried. Zephiran chloride tincture (1:1,000) is applied to the surface of the field with cotton-tipped applicators. Special attention is given to the eyebrow and cilia. The lids are then draped with sterile towels and an eye sheet.

Anesthesia

Local anesthesia was used for 86 of the patients. It was obtained by the injection of 1.0 cc. of procaine hydrochloride and epinephrine hydrochloride into the muscle cone. Hyaluronidase became available for the last 47 cases and was added to the procaine and epinephrine.

I am careful to inject no more than 1.0 cc. of the procaine-epinephrine-hyaluronidase mixture because if 2.0 to 3.0 cc. are used, the eye becomes so soft that the zonular fibers seem to be separated before they actually are, and because the lens loses its resiliency and slips off the erisophake easily. These untoward factors outweigh the superior anesthesia achieved if the larger amount is given.

Akinesia was secured by the Van Lint technique of injecting procaine with epinephrine into the lids. The akinesia was improved in

the last 47 cases by the addition of hyaluronidase to the local anesthesia.

Technique of extraction

The conjunctival flap is limbal-based and is about two mm. wide. Two McLean sutures are preplaced through a limbal groove. The cataract section is made with the von Graefe knife and extended with scissors for half of the limbal circumference. The zonular fibers are separated below by point pressure around the lower limbus with a Budapest lens expressor. This is done before the peripheral iridotomy is performed, or blood is irrigated from the anterior chamber. Contraction of the iris usually begins to occur as soon as either of these steps are taken, and if some of the zonules are already separated, the tumbling and delivery can be done much more quickly.[4]

The peripheral iridotomy is done in the upper iris at the 12-o'clock position. The lens is grasped through the pupillary opening with the Bell erisophake[5] and tumbled, pressure being exerted on the lens from below with the expressor. The assistant holds on to the conjunctival flap at the 12-o'clock position gently and, as the lens is tumbled out, pulls the cornea quickly into place if vitreous loss threatens. The McLean sutures are tied and the conjunctival incision is reunited with a continuous untied suture.

During the progress of this series, certain technical improvements were developed. In the first cases, a Castroviejo cross-action forceps with Arruga tips was used to grasp the lens near its lower edge, and the lens was tumbled. In the last 45 cases, the Bell erisophake has been used, with slipping of the erisophake being unusual and rupture of the capsule in seven cases. Since the "working volume" space is small (0.25 cc.) in this type of erisophake, there is not the danger of aspiration of large amounts of vitreous which can occur with a large suction apparatus.

Summary

The findings are presented of a study of cataract extraction on 103 consecutive one-eyed patients. The three major causes of the loss of the first eye were complications following cataract extraction, primary glaucoma, and severe trauma. As a rule, the complications following cataract extraction in the first eye usually can be avoided in the second, cases of corneal dystrophy being the exception.

Seventy-five percent obtained a visual acuity of 20/40 or better. Those obtaining a lower visual acuity were subjected to careful evaluation, and the findings are tabulated. Glaucoma, corneal dystrophy, and retinal detachment were among the complicating factors.

Critical parts of the preoperative examination and the preparation of the patient, and new developments in the surgical technique are described. The "round-pupil" intracapsular extraction, with a peripheral iridotomy and two corneoscleral sutures, is less likely, in my hands, to be followed by postoperative complications of iris incarceration or prolapse than the extracapsular method combined with iridectomy.

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BORDERLINE FORMS OF GLAUCOMA*

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The conventional classification of glaucoma into primary and secondary types fails to take into account the numerous cases encountered in clinical practice which cannot be classified with absolute certainty either in the one or the other group. This consideration would be only of theoretical interest were it not for the fact that the treatment recommended for secondary glaucoma is often diametrically opposed to that used in primary glaucoma.

In fact, these borderline cases often constitute the most baffling therapeutic problems in the entire field of ophthalmology. From the standpoint of therapy it becomes essential to recognize the existence of cases in which the conventional criteria employed in the classification of the glaucomas do not apply, or at least do not help in the management, each case being treated individually after careful analysis of its clinical features.

Among borderline forms of glaucoma may be mentioned the following types:

1. Some cases of glaucoma following thrombosis of the central retinal vein.
2. Glaucoma associated with retinitis pigmentosa.
3. Glaucoma associated with uncomplicated aphakia.
4. Glaucoma associated with mild iridocyclitis. A most interesting group which falls into this classification is the "syndrome of glaucomatocyclitic crises," described by Posner and Schlossman.[1]

GLAUCOMA IN CENTRAL RETINAL VEIN THROMBOSIS

In several instances of this serious and intractable form of glaucoma, the other eye was found to give a positive Marx and Schmidt water-drinking test. In one case, the other eye subsequently developed chronic simple glaucoma. Moreover, three patients with bilateral primary glaucoma, some years later, developed central vein thrombosis in one eye.

The association of primary glaucoma with central vein thrombosis would appear to be more than coincidental. While attention was called to this relationship repeatedly by Sugar[2] and others, its nature has not thus far been established. The glaucoma may be due to similar sclerotic or thrombotic changes in the minute afferent vessels leading to Schlemm's canal.

These cases usually respond poorly both to miotic therapy and to fistulizing operations. Cyclodiathermy or cycloelectrolysis may be attempted. If this fails, a posterior scleral trephination with fistulization of the vitreous by the method of Lindner may lower the tension and relieve the pain. In an intractable, painful glaucoma, either retrobulbar injection of alcohol or enucleation must be resorted to.

GLAUCOMA AND RETINITIS PIGMENTOSA

I have observed three cases of glaucoma associated with retinitis pigmentosa. Two of the patients had bilateral acute congestive glaucoma, the other patient suffered from chronic simple glaucoma. In all cases the reaction to medication and surgery was like that in primary glaucoma. Clinically, the retinitis pigmentosa did not appear to be causally related.

In cases in which the eyes were examined histologically, Gartner and Schlossman[3] found an open canal of Schlemm and no increase of pigment in the trabeculum. In some instances at least, the association of the two diseases may have a genetic basis. One of the patients with congestive glaucoma and retinitis pigmentosa has one sis-

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ter who has the same two diseases, while another sister has only retinitis pigmentosa. Blessig[4] reports a sibship of nine members of whom two sisters had chronic simple glaucoma, while two brothers and one sister had pigmentary degeneration without glaucoma.

Glaucoma associated with uncomplicated aphakia

It is always both a puzzling experience and a sad disappointment to find a chronic glaucoma developing some weeks or months after a well-executed cataract extraction.

If one eliminates from consideration those cases of obviously secondary glaucoma which are caused by some type of surgical complication—such as delayed reformation of the anterior chamber, loss of vitreous, prolapse of iris, or postoperative uveitis—there still remain instances in which there are no postoperative complications or abnormal findings present which could satisfactorily account for the increased tension.

While clinical experience leaves no room for doubt that the cataract extraction is at least a precipitating factor in the causation of the glaucoma, its etiologic role is still obscure.

On clinical considerations alone, this type of aphakic glaucoma appears to be either primary or closely related to it. The tension becomes progressively more elevated, excavation and atrophy of the optic disc result in typical field changes, yet the patient experiences no subjective symptoms. Usually, the anterior chamber is deep and the angle wide. Gonioscopic findings show no consistent deviations from the normal. A herniation of the vitreous, if present, is probably of no pathogenetic significance. Even if strands of vitreous are seen to pervade the aqueous in the anterior chamber, it is doubtful whether they can be held accountable for the glaucoma; for I have seen vitreous in the anterior chambers of aphakic eyes with normal tension.

It is important to bear in mind that both senile cataract and glaucoma occur in the older age group. It may even be that both diseases owe their genesis to similar alterations in the permeability of the cuticular membranes of the eye. Cataract may be caused by changes in the lens capsule, while chronic simple glaucoma is probably due to altered permeability of the cuticular membrane lining the trabecular spaces.

In several cases, I have observed a reciprocal relationship to exist between cataract and chronic simple glaucoma. The eye which had the glaucoma retained good central vision and a clear lens for several years. The fellow eye which was free from glaucoma, even to the extent of giving negative provocative tests, developed a rapidly progressive cataract. Intracapsular extraction of the cataract in three patients resulted in normal vision and field. In one patient the tension remained normal for at least four years. In the other two patients the tension of the aphakic eye later became elevated but could easily be controlled by weak solutions of pilocarpine.

I still am mystified by the occurrence of this apparent reciprocity between glaucoma and cataract in the same patient. I could venture an explanation which, for the present, is merely an hypothesis without actual proof.

It is a well-known fact that the formation of cataract in glaucoma may be hastened by any surgical procedure which results in a marked lowering of the tension. This is particularly true of the Elliot trephining operation which is frequently followed by hypotension.

It has been shown by various workers, such as Goldmann and Bárány, using different experimental methods, that the rate of flow of aqueous out of the eyeball is constant in the normal eye and that it tends to remain constant regardless of changes in the intraocular pressure. Bárány even goes so far as to assert that there must be a controlling mechanism to maintain this constancy of the rate of aqueous flow, since the aque-

Fig. 1 (Posner). Section through scleral cataract incision, showing closure of angle by scarring of trabeculum. Patient died of coronary thrombosis within several weeks of the operation. Tension remained normal during this period of time.

ous probably plays a role in the metabolism of the lens.

In glaucoma simplex, the elevation of the intraocular pressure represents an attempt to compensate for the impaired facility of outflow of the aqueous through the trabeculum. If it is assumed that both eyes suffer the same impairment of aqueous outflow, but only one eye shows a corresponding rise of the intraocular pressure, the other eye with normal tension is likely to have a less efficient aqueous circulation and hence would be prone to develop a cataract.

Interference with the drainage of aqueous through the trabeculum by the scarring of the cataract incision may be another important cause of aphakic glaucoma. The present-day trend toward intracapsular extractions has led many surgeons to place the incision behind the limbus, so as to obtain a larger opening. Such an incision must necessarily pass through the corneoscleral trabeculum and the resultant scarring will thus impair the drainage of aqueous through at least one half of the circumference of the angle.

In a normal eye, enough trabeculum would still be left to maintain a normal aqueous circulation. But if the permeability of the trabeculum has previously been reduced, then so little reserve capacity may have remained that the loss of 50 percent of its function will result in inadequate drainage with consequent glaucoma.

Figure 1 is a section through a scleral incision showing the closure of the angle by scarring after an intracapsular cataract extraction. The tension remained normal until the patient died a few weeks later of a coronary thrombosis. Figure 2 is a section through the lower half of the same eyeball, showing a normal angle.

In view of the existence of these indicated relationships between glaucoma and cataract, the most important therapeutic measures are in the nature of prophylaxis.

It is advisable to place the cataract incision as close to the limbus as possible, thus minimizing the scarring of the trabeculum. By cutting the cornea perpendicularly with scissors, less shelving of the wound will result and the opening will usually be adequate.

The tension should always be measured before operating for cataract. If it is high normal, a provocative test (preferably the water-drinking test of Marx and Schmidt) should be carried out. In any questionable case, atropine should not be used subsequent to the first instillation made at the time of the operation. If a postoperative iritis develops, one can use homatropine or neosynephrine. The prolonged routine use of atropine in older persons is inadvisable even

Fig. 2 (Posner). Section through lower half of same eyeball, showing that angle was normal beyond the line of incision. The retention of one half of the normal trabeculum explains the absence of glaucoma in this case.

if no definite contraindication exists.

If aphakic glaucoma has occurred in one eye, a provocative test should be done on the other eye prior to operation, and the above rules regarding the use of mydriatics should be followed—even if the provocative test is negative.

The treatment of aphakic glaucoma is similar to that of chronic simple glaucoma. Cases which prove refractory to pilocarpine can often be controlled by the more powerful cholinesterase-inhibiting drugs, such as DFP.

When medical treatment fails, surgery must be resorted to. Technically, the most feasible procedure is a cyclodialysis. I have been following the method as originally described by Heine. This has given me generally satisfactory results.

The conjunctiva is incised either below and temporally or above and temporally, five mm. from the limbus, and the sclera is exposed. A three-mm. scleral incision is made parallel to and four mm. from the limbus.

A spatula (preferably the one of Elschnig) is inserted through the incision toward the anterior chamber, hugging the sclera. When the end of the spatula has reached the pupillary area, the spatula is rotated by 90 degrees first in one, then in the other direction—always keeping it in contact with the sclera —and is then withdrawn, preferably in the position which it had when originally inserted. The conjunctival incision only is sutured. Neither a miotic nor a mydriatic is used.

An operation which is gaining in popularity is cyclodiathermy.

If the anterior chamber is deep and there is no herniation or prolapse of the vitreous, an Elliot trephining operation can be performed.

Glaucoma associated with iridocyclitis

When glaucoma develops in the course of an active iridocyclitis, it nearly always presents a serious and baffling therapeutic problem. Even though, by the usual diagnostic criteria, the glaucoma must be classified as secondary, the capriciousness of its response to treatment often leads one to doubt the

validity of any hard and fast classification. The glaucoma frequently fails to respond to mydriatics, while it may show at least temporary improvement with miotics. Attempts to find the etiologic factors responsible for the iridocyclitis and glaucoma are usually disappointing. Even if some foci of infection or other pathologic states are uncovered, such findings usually prove of little value in the medical control of the ocular process.

Each case must be dealt with individually. Both mydriatics and miotics should be used with caution, the dosage being determined not only by the height of the tension or the prominence of inflammatory signs, but also by the functional tolerance which the eye shows toward the pathologic process.

Fortunately, in many of these cases, the eye will tolerate high tension for prolonged periods without showing either loss of field or cupping of the optic nerve. This may be accounted for, at least in part, by the wide diurnal fluctuations of the ocular tension. Patients often volunteer the information that their vision clears and the pain subsides during a certain part of the day, usually in the evening. The periodic remission of the tension and the lessening of the corneal edema can be verified if the patient is examined at various times of the day.

In the treatment of glaucoma associated with active iridocyclitis, pilocarpine (1.0 to 2.0 percent) may have to be alternated with such mydriatics as homatropine (2.0 percent) or neosynephrine (10 percent), so as to forestall the formation of dense posterior synechias. The inflammatory process can often be brought under control by the use of cortisone both systemically and locally. This may either exert a directly favorable influence on the glaucoma, or at least facilitate the employment of miotics without aggravating the iridocyclitis or causing excessive pain.

With regard to prognosis, it has been my experience that under judicious medical treatment and careful observation, no loss of visual field need be feared even if the tension has been elevated for several weeks.

If the field should show evidence of impairment, an iridectomy or an iridosclerectomy may have to be performed. The common practice of repeated paracenteses is, I believe, to be deprecated since it is based on the false premise that the elevation of tension, per se, is deleterious to the function of the eye. From my experience with cases which have recovered spontaneously, I am convinced that those instances in which paracentesis is followed by a cure do not necessarily establish a cause-and-effect relationship.

On the other hand, there are other cases in which repeated paracenteses are actually detrimental. The evacuation of aqueous leads to the formation of peripheral anterior synechias, especially in view of the plasmoid nature of the aqueous and of the adhesive quality of the endothelial lining of the collapsed anterior chamber, which is evidenced by the formation of keratic precipitates. The use of paracenteses in certain cases may thus lead to structural changes in the angle which result in an irreversible form of secondary glaucoma, refractory to all types of treatment, including surgery.

Syndrome of glaucomatocyclitic crises

This is a special type of recurrent acute unilateral glaucoma associated with mild cyclitis. Since its first description by Posner and Schlossman in 1948[1] numerous cases of this syndrome have been encountered by these and other observers.[5–9]

The clinical features which characterize this clinical entity are given in the following summary which is taken from the original paper,[1] with slight modifications:

1. The disease is unilateral and the same eye is always affected. If anisocoria is present, the affected eye has the wider pupil.

2. The presenting symptoms are slight discomfort, colored halos, and blurring of vision. However, there is no pain and the vision is normal or almost normal, even at the height of the attack.

3. The eye is white since there is no ciliary injection, although there may be slight injection of the bulbar conjunctiva.

4. The ocular hypertension may appear a day or so before, or simultaneously with cells in the aqueous. A few small discrete, unpigmented keratic precipitates appear a few hours to two to three days later. The tension which may reach 90 mm. Hg (Schiøtz) during the attack may return to normal within a day or so and no attack has been known to last for more than two weeks. The end of the attack is usually signalled by a temporary drop of the tension to a subnormal level. The keratic precipitates gradually fade and disappear completely within one month. They may be entirely absent during some attacks.

5. Posterior synechias do not form; there is never more than a trace of aqueous flare, and the pupil is wider than its fellow; all characteristics which distinguish it from the typical acute iridocyclitis.

6. The anterior chamber is not shallow; the angle is not narrow and remains open during the attack.

7. The attacks recur with varying frequency and without any apparent cause. A history of previous similar attacks can frequently be obtained from the patient.

8. No permanent impairment of the visual acuity or visual field is found and no cupping of the disc results over a period of several years in spite of recurrent attacks.

9. During the intervals, the tension is normal, and provocative tests for glaucoma give normal results.

10. The attack subsides spontaneously. Treatment does not seem to shorten the total duration of the attack, although it may lower the tension temporarily. Mild miotics, such as pilocarpine (0.25 to 1.0 percent) may be alternated with neosynephrine (10 percent). Stronger miotics should not be used as they tend to give rise to pain and congestion. Topical cortisone is often helpful in allaying the cyclitis.

11. Surgical procedures do not prevent recurrences. Paracentesis may help lower the tension, but is not necessary since no permanent damage results from the elevated tension.

Case reports

The following brief résumés of actual cases will serve to depict the clinical course of this syndrome in a more graphic manner.

Case 1

A 55-year-old woman began to have recurrent attacks of blurred vision of her right eye as long as 24 years ago. During one such attack, a prominent ophthalmologist made the diagnosis of glaucoma and prescribed pilocarpine hydrochloride (1.0 percent). The attack subsided after a few days.

While using the miotic prophylactically, she had another attack in the same eye. Another ophthalmologist discovered a few keratic precipitates and ordered homatropine. Again, the attack subsided within a few days.

After suffering a few more attacks in the course of a few years, which were treated successfully either with homatropine or pilocarpine, depending on whether the ophthalmologist she consulted used the slitlamp or not, she was symptom-free until 1947, when she had a rather severe attack of glaucoma with tension as high as 90 mm. Hg. A few fresh keratic precipitates were noted on the third day of the attack. She was kept comfortable with pilocarpine hydrochloride (0.25 percent) until the tension returned to normal within two weeks. The precipitates disappeared within one month. There were no posterior synechias, the angle was wide and remained open, and the field remained normal.

Case 2

A 46-year-old woman had two attacks within one year of blurring of vision and "itching" of her right eye. On her second attack an ophthalmologist found a white eye, no keratic precipitates, a slightly dilated pupil, and a tension of 70 mm. Hg (Schiøtz). He instilled one drop of eserine, after which the eye became painful, red, and tender.

When I saw her the following day, the tension was 77 mm. Hg, the pupil contracted, and the slitlamp showed a few fresh keratic precipitates. The right iris was gray, whereas the left iris was brown. The vision was 20/20. The angle was moderately narrow, but open. She received no treatment except for cold compresses, yet the pain subsided promptly, the tension returned to normal within 15 days, and the precipitates disappeared within three weeks.

Case 3

This 31-year-old man, a patient of Dr. A. John Reinhorn, had his first attack of blurred vision and colored halos in his right eye 10 years ago. The attack subsided within two to three days under

pilocarpine treatment. One year later, he had a recurrence which responded similarly.

When I saw him, he complained of fogginess and colored halos of two days' duration. He gave no history of allergy except for occasional urticaria after eating spicy foods. His right pupil was somewhat wider than the left and he thought that it had always been so, at least since the time of his first attack. The corrected vision was 20/20 in each eye. The anterior chambers were deep and the angles wide. There was no cupping. Six small keratic precipitates were seen in the right eye. The left eye was normal. The tension was R.E., 49 mm. Hg (Schiøtz); L.E. 20 mm. Hg.

Under cortisone drops and pilocarpine hydrochloride (1.0 percent) the tension returned to normal after two days and the precipitates disappeared within one week.

As noted, cases belonging to this syndrome are more likely to be confused with primary than with secondary glaucoma. Any case of acute glaucoma in which the eye does not have a narrow angle, or in which the angle appears to be open, should be watched by daily slitlamp studies for the appearance of keratic precipitates.

Until the diagnosis is established, treatment should be limited to weak miotics provided that the visual field and acuity show no evidence of progressive impairment.

This syndrome derives added importance from the fact that it represents a borderline form of glaucoma between the primary and the secondary, since there undoubtedly are cases diagnosed as primary glaucoma which resemble this syndrome in all respects except for the presence of keratic precipitates.

Summary

Some cases of glaucoma cannot be classified with certainty either as primary or secondary glaucoma. From the standpoint of therapy, it becomes essential to recognize the existence of cases in which the conventional criteria employed in the classification of the glaucomas do not apply, or at least do not help in the management, each case being treated individually after careful analysis of its clinical features.

Among these borderline forms of glaucomas are: (1) Some cases of glaucoma following thrombosis of the central retinal vein; (2) glaucoma associated with retinitis pigmentosa; (3) glaucoma associated with uncomplicated aphakia; (4) glaucoma associated with mild iridocyclitis, particularly the "syndrome of glaucomatocyclitic crises" (Posner and Schlossman).

The clinical aspects and the treatment of these forms of glaucoma are discussed, emphasis being placed on the last two groups.

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EXPERIMENTAL AND CLINICAL OBSERVATIONS OF THE EFFECTS OF CORTISONE IN CORNEAL LESIONS*

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During the past year the value of cortisone in the treatment of a great number of eye conditions has become well established. This has been largely through the clinical use of the adrenal cortical steroid hormones which have been administered parenterally, topically, and by subconjunctival injection. The topical use of cortisone has been most effective in lesions of the anterior segment of the eyeball. We wish to present our observations on the effect of subconjunctival injections of cortisone acetate on wound healing in the corneas of rabbits and to correlate our findings with the topical use of cortisone in a variety of corneal lesions in patients.

Fig. 1 (James, Power, and Ripple). One-week control: catgut suture in conjunctiva. (Typical foreign-body reaction.)

Fig. 2 (James, Power, and Ripple). Two-week control: catgut suture in conjunctiva. (Marked reaction and dissolution of gut.)

The effects of cortisone on ocular wounds can be anticipated to some extent by the work of others, who found its chief action to be an inhibition of mesenchymal growth (granulation tissue) and the shrinkage of collagenous tissue. Plotz and others[1] described wounds of rabbit ears in which the epithelium grew down into the wound over bare adipose tissue because of the lack of ground substance. Howes and his co-workers[2] noted the same with rats; Spain and his collaborators[3] with mice, and Creditor and his associates[4] cited two human cases under ACTH therapy in which wound healing was very much delayed.

Plotz[5] noted delayed fracture and intestinal wound healing. Most workers found little effect on epithelial healing, although in some cases the epithelium was described as being thinner and composed of smaller cells. Castor and Baker[6] noted a thinning of dermis, cessation of hair growth, and shrinkage of the sebaceous glands with the local appli-

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† By invitation.

cation of cortisone. Jones and Meyer[7] induced lye burns in rabbit corneas by injecting solutions of sodium hydroxide into the stroma and noted a marked tendency for cortisone to inhibit subsequent vascularization.

The action of cortisone in inhibiting mesenchymal healing is closely related to its beneficial results in the treatment of inflammatory disease which involves collagenous tissues. Plotz[1] calls this action a delay in host

Fig. 3 (James, Power, and Ripple). Two-week cortisone: catgut in conjunctiva. (No foreign-body reaction or dissolution of gut.)

activity. The mechanism is believed due to an inhibition of hyaluronidase which increases tissue permeability (spreading factor). Seifter, Baeder, and Begany[8] using synovial membrane and Seifter, Baeder and Dervines[9] using bladder membrane noted that cortisone decreased permeability. Howes[2] found a decrease in hyaluronidase in wounds of cortisone-treated rats, but Creditor[4] could not demonstrate improved wound healing in an ACTH-treated patient when his wound was irrigated with hyaluronidase.

The early closure of a corneal wound is accomplished by swelling and adhesion of the

Fig. 4 (James, Power, and Ripple). One-week control: partial keratectomy. (Epithelial and early stromal reaction.)

corneal stroma with mechanical and proliferative spread of the epithelium. Keratoblasts (corneal corpuscles) wander in from the near-by stroma, bind the edges, and fill the anterior and posterior gaps. Endothelium grows over the granulating posterior gap and

Fig. 5 (James, Power, and Ripple). Two-week control: partial keratectomy. (Granulation tissue and cellular infiltration.)

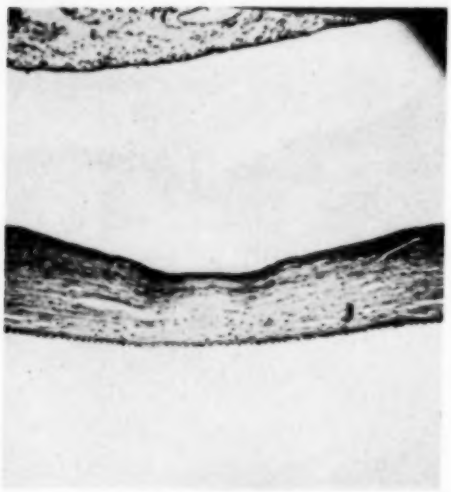

Fig. 6 (James, Power, and Ripple). Two-week cortisone: partial keratectomy. (Shrunken fibers and no stromal reaction.)

finally a scar forms which produces a firm wound. In view of the prominent role that the mesenchymal tissues take in repair it would be expected that cortisone would influence healing.

PROCEDURE

To observe the effect of cortisone on ocular wounds the following procedures were carried out in 17 rabbits; a total of 34 eyes.

1. A conjunctival incision one cm. long parallel to the limbus at the site of the insertion of the superior rectus muscle was made with a scissors and closed with 3-0 plain catgut.

2. Corneal section five mm. to seven mm. long was made with a Graefe knife in clear cornea midway between the center of the cornea and the limbus. A preplaced 6-0 black silk suture was tightened and tied.

3. A two-mm. trephine button of tissue was removed from the lower central quadrant of the cornea to a depth of one half the corneal thickness.

Eight rabbits served as controls; four being killed in one week and the other four in two weeks, after which their eyes were enucleated. The other group of nine rabbits received a subconjunctival injection of 6.0 mg. (0.25 cc.) of standard cortisone acetate (Merck) every other day in each eye. Four of these rabbits were killed in a week while the other five were killed at two weeks and their eyes enucleated. Microscopic sections were made and studied.

Conjunctival section. The difference between the two groups was marked in this study because of the inhibition of inflammatory response about the catgut in the cortisone-treated eyes. The control eyes showed the typical foreign body reaction with a marked infiltration of leukocytes and edema with partial destruction of the catgut suture (figs. 1 and 2). The cortisone-treated eyes did not show this typical response, and the sections revealed the intact gut lying inert in the subconjunctival tissue (fig. 3). Even at two weeks there was practically no cellular infiltration and no sign of deterioration of the gut.

Superficial keratectomy. A study of these wounds demonstrated marked differences in the healing process. In one week the control eyes showed a good filling of the defect of

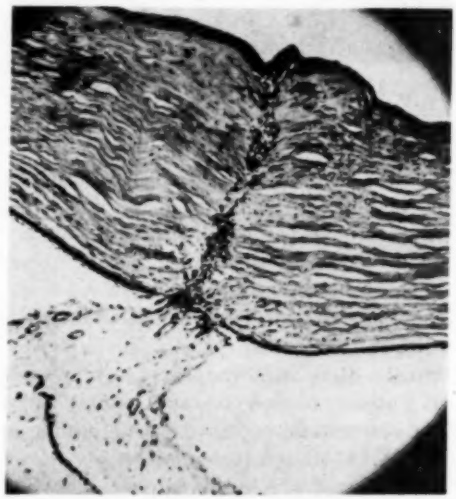

Fig. 7 (James, Power, and Ripple). One-week control: corneal section. (Typical healing reaction.)

epithelium, while the stroma was beginning to respond with migration of keratoblasts to the base of the wound (fig. 4). The two-week controls had a generous bed of granulation tissue at the base of the wound while the surrounding stroma was well infiltrated with round cells (fig. 5).

The cortisone-treated eyes, on the other hand, demonstrated a thinner epithelium with slightly smaller cells but there appeared to be no delay in the epithelization. This epithelium was not so firmly attached to the base

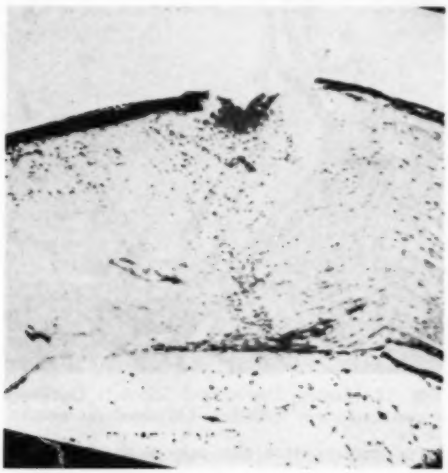

Fig. 8 (James, Power, and Ripple). Two-week control: corneal section. (Typical healing reaction.)

of the wound and was actually detached on some of the sections. The stroma showed a greater difference in its reaction, with shrinkage of the corneal lamellas at the base of the wound; lack of wandering keratoblasts and granulation tissue, and lack of surrounding cellular infiltration (figs. 6 and 7). Leopold and his co-workers[10] undertook a similar study and noted a delay in granulation tissue and in epithelial growth which, as evidence by staining, lasted twice as long in the eyes treated by cortisone.

Corneal section. The microscopic study of the corneal sections displayed the greatest number of differential points of interest between the control and the cortisone-treated

Fig. 9 (James, Power, and Ripple). One-week cortisone: corneal section. (Few keratoblasts in gaping wound. Stroma inert with no infiltration. Epithelium normal.)

animals. These differences occurred with regularity in spite of the variable surgical results following a uniform surgical procedure.

The control eyes demonstrated the normal healing reaction described earlier. There was

Fig. 10 (James, Power, and Ripple). Two-week cortisone: corneal section. (Same as Figure 9 but with very wide gaping posterior wound. No endothelial regeneration.)

Fig. 11 (James, Power, and Ripple). One-week cortisone: silk suture track. (Lack of reaction.)

a marked cellular infiltration with good healing of the wound (figs. 8 and 9).

In the animals treated by cortisone the epithelium, as was the case in the superficial keratectomy wounds, showed no delay and little alteration from the normal healing response as seen in the controls. The epithelium was seen to cover all wounds. The cortisone-treated eyes in general showed very poor healing even at two weeks. Notably there was a marked deficiency in keratoblasts and thus granulation tissue (fig. 10). The wound edges remained separated and appeared as if they had just been cut and the surrounding stroma did not show the typical cellular infiltration. The posterior gap especially remained widely separated as compared to the anterior defect which was bridged by epithelium. There was no evidence of regeneration of endothelium (fig. 11). In one section (fig. 12) of a cortisone-treated eye a black silk suture track was seen and, as was the case with the catgut suture, there was practically no foreign body response. An iris incarceration in another section of a cortisone-treated animal showed the same lack of reaction in the corneal stroma.

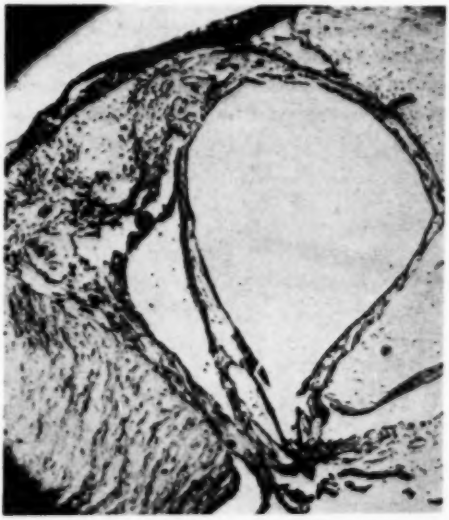

Fig. 12 (James, Power, and Ripple). One-week cortisone: iris inclusion. (Minimal reaction.)

TABLE 1

PROCEDURE AND RESULTS

Procedure	Control Group (16 eyes)	Cortisone Group (18 eyes)
Conjunctival wound closed with 3-0 catgut	Foreign body reaction with cellular infiltration and edema. Dissolution of gut	Greatly suppressed foreign body reaction with no dissolution of gut
Partial keratectomy	Typical growth of epithelium on a granulating bed of new mesenchymal tissue with surrounding cellular infiltration	Epithelium thinner and composed of smaller cells which covered shrinking corneal lamellas with no granulation tissue or cellular infiltration
Corneal section closed with 6-0 black silk	Typical healing reaction with regeneration of epithelium and endothelium and migration of keratoblasts to wound-binding edges. Cellular infiltration of surrounding stroma	Greatly suppressed healing. Epithelium fairly normal but posterior wound gaped widely: no endothelium and a marked reduction in keratoblasts and other cellular infiltration

TABLE 2

RESULTS OF INSTILLATION OF CORTISONE ACETATE

Diagnosis	Cases	Drug	Improvement	No Change	Comment
Glaucoma secondary Corneal edema	1	Cortisone q.2.h.	Marked		Halos and corneal edema cleared
Corneal dystrophy (early)	1	Cortisone q.2.h. to t.i.d.	Subjective	No objective corneal change	Relief of halos and blurred vision
Corneal dystrophy (far advanced)	2	Cortisone q.4.h.	Subjective	No objective corneal change	Patients were more comfortable
Corneal abrasions	6	Cortisone q.2.h.	Excellent		Pain relieved. Three injuries were fingernail scratches, three from evergreen needles
Recurrent erosion	4	Cortisone q.2.h.	Excellent		Pain relieved. Stromal reaction cleared
Punctate keratitis	2	Cortisone q.1.h.	1 improved	1 did not improve	Vague subjective response
Lime burn	1	Cortisone q.2.h.	Excellent		Opaque cornea cleared with no vascularization. Visual acuity 6/9
Sclerosing keratitis	3	Cortisone q.2.h.	Excellent		Inflammatory changes and growth of vessels subsided
Herpes zoster of cornea	2	Cortisone q.2.h.	Excellent		Inflammatory symptoms and corneal edema improved. Less scarring
Dendritic ulcer	4	Cortisone	Slight		Pain lessened. Inflammation less. Lesions only healed after application of iodine
Acna rosacea keratitis	2	Cortisone q.2.h.	Excellent		Pain subsided. Inflammatory signs and vascularization of cornea decreased
Keratitis, ulcerative with trachoma	2	Cortisone q.2.h.	Excellent		Pain subsided. Inflammatory signs and vascularization of cornea decrease
Total	30		26	4	

Table 1 summarizes these observations.

Table 2 summarizes our experience with the instillation of cortisone acetate in a 1:4 saline suspension in the human eye.

COMMENT

The efficacy of cortisone in lesions of the anterior segment of the eye when used locally by injection or by drop instillation is well established clinically by many reports in the literature. The additional advantages of local use are: (1) The avoidance of general systemic reactions, (2) the elimination of hospitalization of the patient, (3) the lowered cost of the local administration of cortisone, and (4) the long period of treatment.

The surgical wounds of the rabbit eyes were affected by cortisone in the following manner. (1) There was a slight effect on the surface epithelium of the cornea and conjunctiva. (2) The tissue reaction of the corneal stroma fibroblastic proliferation, and lymphocytic infiltrations, was retarded by subconjunctival injections of cortisone. (3) The foreign body reaction about the silk and gut sutures was slight in comparison with the control eyes. (4) Existing stroma at the base of the partial keratectomy was shrunken.

From the results of this study it can be said that cortisone may have a role in ophthalmic surgery if it is desirable to keep mesenchymal healing at a minimum, as in filtration procedures, partial keratectomies, pterygium, and lacrimal duct operations. Scheie and his collaborators[11] treated nine postoperative corneal transplants with either ACTH or cortisone and reported somewhat encouraging results, Cortisone, however, would be contraindicated where sound wound healing is essential, as in cataract surgery.

In the corneal lesions treated by instillation of a 1:4 saline dilution of cortisone acetate (Merck) the most favorable results were obtained in superficial traumatic abrasions before infection or prolonged treatment with local anesthetics had occurred. From the clinical findings as seen under the biomicroscope we believe that the epithelization of the cornea is not hindered by cortisone. Rather, the lack of stromal activity in certain inflammations favors early coverage of the wounds. The reaction about deep-seated herpetic lesions was minimized. The retardation of corneal vascularization was noted. From the patient's viewpoint the decrease in ocular pain was most welcome.

Conclusion

In superficial or nonpenetrating lesions of the cornea cortisone may be used without danger and with reasonable expectation of minimizing the subsequent scarring and vascularization. Our experience with ulcerative herpetic lesions has been favorable although ultimate cauterization with tincture of iodine was necessary for healing. Wounds of the globe exposing the intraocular contents of the eye should not be treated with cortisone if firm union of the wound edges is desired.

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Ophthalmic Miniature

With respect to the number of blind, we found from the inquiry instituted in 1851 that they amounted to 7,587, or one in every 864 of the population, the sexes being in proportion of 100 males to 111.45 females.

W. R. Wilde, "Report on the number, sexes, and condition of the blind in Ireland," *Medical Times*, London, 1862.

HISTORIC AND ETHNOLOGIC FACTORS IN THE DISTRIBUTION OF TRACHOMA

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Introduction

It is well known that contagious diseases are not equally distributed in different countries and among different peoples. There are many reasons—some of them known and others still unknown—for this inequality.

In addition to the properties of the disease and its agents (such as its being acute or chronic; spread directly or indirectly through living beings; giving or not giving immunity; and so forth), there are also geographic (climatic and geologic) factors, as well as cultural, social, and, especially, historic ones. Only an accurate knowledge of all these items can give us a basis which would enable us to know and to understand the problems of epidemiology in general and those of trachoma in particular.

As the exact study of contagious diseases dates from the last century only, little information about their extension all over the world is to be obtained from former medical literature. It is, for example, impossible to know when epidemics of influenza, typhoid, spotted typhus, and pest began or to tell which diseases were frequent in former times and which did not exist at all.

Dr. Stock thinks that, since whooping cough was never mentioned until 1557, it is a proof that this typical disease had not existed before then. I cannot agree with this opinion.

Since pannus, for instance, never was mentioned in ancient literature, many investigators came to the conclusion that the Arabs were the first to notice this phenomenon. But can it be imagined that, in days of old, no pannus accompanied trachoma; or that the Greeks, who left us many excellent pictures of this illness, took no notice of corneal inflammation? As a matter of fact, Meyerhof did find a description of this inflammation, under the name of chirsophdanmia.

On the other hand, it is impossible to understand the present state of contagious diseases without knowing their evolution and historic background. This makes the study of these problems rather difficult. If the literature does not contain a sufficient number of facts, we have to consider all sorts of indirect historic testimonies, conjectures, and more or less acceptable hypotheses.

All these factors concern contagious diseases in general and trachoma in particular.

Geographic distribution

When we consider the map of the world[1] from the point of view of the extension of trachoma, we at once come across many striking singularities:

A. *In the Baltic-Sea zone,* we observe that the countries of the eastern and southern coasts of the sea are all, more or less, afflicted by trachoma—Finland, Esthonia, Latvia, Lithuania, Eastern Prussia, and Pomerania; while the countries lying on the western coast are nearly free from trachoma—Sweden, Norway, Denmark.

B. *In the center of the immense spaces of European Russia,* there are regions, such as the Kaluga district, about which I, myself, can testify as to their being exempt from trachoma; whereas, the adjacent regions are more or less afflicted by the disease.

C. It has long ago been noticed that in *Central Equatorial Africa* the Negroes are not subject to trachoma; only those living in Egypt, Senegal, and Brazil suffer from the disease, although in a smaller proportion than the people among whom they dwell.

What caused this state of things in these and in many other analogical cases?

Historic factors

As long ago as 1927, at an ophthalmologists' meeting in Jerusalem, I expressed my opinion as to the cause of these phenomena, which is, to my mind, a historic one.

Where and when did trachoma originate? This is the first question with which we have to deal.

We do not know when and where most diseases appeared for the first time. Some—for example, tuberculosis, diphtheria, poliomyelitis—we believe to have originated simultaneously in several places, noticing that their distribution is almost equal in various corners of the world. Cholera, on the other hand, came originally from India, whence it spread, by sea and land, all over the world.

Trachoma is mentioned in the oldest known documents: in Egyptian papyri (*Papyrus Ebers*); in Greek literature (Herodotus, Aristophanes, Hippocrates); hints can be found in the Bible. It may be supposed that trachoma originated in the days of old when humanity lived in separate tribes, before states and nations had been formed. It may be further supposed that trachoma appeared in a few tribes only, while other tribes were spared.

One of the places where trachoma originated, according to Dr. MacCallan, and in my opinion as well, is Central Asia, for all the tribes coming from those regions (the Mongolian, Mongolo-Tartar and Ugro-Finnic) are severely afflicted with the disease. Not a single one of these races is spared by trachoma.

All the Finnic tribes (Finns, Tshuvashes, Esthonians, Letts), as well as Magyars whose language is akin to that of the Finns, suffer from trachoma more than the neighboring peoples among whom they live. This is also the case of other Mongolian tribes—the Tartars, Kalmucks, Bashkirs, Chinese, Japanese—all of which are afflicted with trachoma. All these tribes in their migrations transmitted the disease to other nations, which had not known trachoma; and nearly always people who contracted the disease suffered from it less than those who propagated it.

Since German and Slavic nations did not know trachoma, if, in the course of their history, they did not come into contact with nations subject to trachoma, they remained free from the disease. This is also the reason why certain regions in Central Russia, in which the agricultural population has no wide commercial intercourse with the rest of the country, are almost free from trachoma. In other districts, however, bordering upon regions inhabited by Finns, Mongols, or Lithuanians, the rate of trachoma is from one to five percent.

While among Mongolians (Tartars, Bashkirs, and so forth) and Finns (Mari, Tshuvashes, and so forth) the rate of trachoma was 30 percent in North Russia and 50 percent in South Russia, among the neighboring Russian population, the rate of the disease never went above five or six percent.

The Poles, being a Slavic race like the Russians, suffer little from trachoma (an incidence of 1.0 to 1.5 percent). To my mind, the source of their disease lies in Lithuania. Lithuania, a small country in the Niemen basin, has played an important part in the diffusion of trachoma among the neighboring nations. There is no doubt that the Lithuanians, whose language is very much akin to Sanskrit, the language of ancient India, had dwelt in prehistoric days near the Himalayas, whence they had migrated, by way of Turkestan or Caucasus to the vast Scythian plains and thence to the Baltic Sea.

Medical statistics show a much higher rate of trachoma among the Lithuanians than among the neighboring peoples. Avijonis reported 30 to 35 percent of trachoma and Rostovsky 15 percent in the district of Vilna. Among the Poles, on the other hand, the rate of trachoma is 1.0 to 1.5 percent; in East Prussia, three to four percent, and in Latvia, four percent. In White Russia, which was for a long period under Lithuanian domination, the rate of trachoma is much higher than among the Poles. Dobreizer

places White Russia second in the geographic incidence of trachoma in Soviet Russia, where, in 1925, there were 357,412 persons suffering from trachoma.

The German race, like the Slavic, is not afflicted by trachoma. The disease scarcely exists in Central Germany. Only in Eastern Prussia, owing to contact with Lithuania, is there a certain percentage of trachoma.

In Germany, itself, there still exist small localities where the contagion is stronger, such as the Rhine district and Westphalia; this is accounted for by a historic moment, the importance of which is unanimously recognized, namely the Napoleonic wars. The French troops concentrated in these districts after their return from Egypt where they had contracted the Egyptian trachoma.

It is only natural that Swedes, Danes, Norwegians and English are free from trachoma.

Great historic events connected with mass migrations, such as migration of peoples in great wars (Crusades, Arabian conquests, Mongolian wars, Napoleonic wars, the Civil War in Russia in 1917 to 1920), play a leading role in the spread of epidemics in general and of trachoma in particular.

It is well known that during the Crusades leprosy was introduced into Europe; there is no information, however, as to trachoma. And yet all the peoples of the Near East—Syria, Palestine, Mesopotamia, and Egypt—were already affflicted with trachoma, owing mainly to the conquest of these countries by the Arabs.

In the middle of the seventh century A.D., the followers of Mohammed left the Arabian peninsula with the object of conquering the world. In a short period of time they succeeded in getting hold of the spaces between Spain and India and reached the Sunda Islands.

Now, wherever there is an Arab-mixed population, the Arabs present a much higher percentage of trachoma than their neighbors. For example, in the Java Islands, according to Beeker, the Arabs suffer from trachoma much more than the Malayans, in spite of the latters' lower economic and cultural level. The same can be observed in Syria; the Moslems are more afflicted by trachoma than the Christians.

It is more than probable that trachoma originated among the Arabs who spread it from India to Ethiopia and from Egypt to Spain, all through North Africa.

In Egypt which is now considered as a source of trachoma, the disease formerly was not very frequent. At least, judging from what is to be found in ancient literature (Hirschberg), there is no reason to suppose that Egypt was heavily infected with trachoma. It was only after the Arab conquest that the disease spread to such an extent that the Egyptian government had to establish, as far back as in the eighth century, a special hospital for diseases of the eye—to my knowledge, the first of this kind.

In Spain, it is mainly the seashore towns which are infected with trachoma—so much so that, at the International Congress of 1928, Nicholich expressed the opinion that the trachoma agent is to be found in the sea. This opinion has not been corroborated by any proofs. The truth is that Spanish seashore towns, during the 700 years of Arab domination, were in constant contact with North Africa and the Middle East, which accounts for trachoma spreading to them.

During the last few decades, we have witnessed the spread of trachoma from North Africa to Equatorial Africa. As I have already stated, the Negroes were free from trachoma until they came under the control of the French, who built roads and developed ways of communication between Algeria and Morocco, on the one hand, and Senegambia and Central Africa, on the other, and contributed to the growth of intercourse, mostly commercial, between these countries.

All the physicians who worked in Central Africa noticed that trachoma spread from North to South in regions where Arabs and Berbers of northern origin had settled. The same thing happened in Kamarun after the

first World War, when this country had been annexed by the French.

The Mongolian invasion of Russia by Tartars in 1240-41 brought a new wave of trachoma. Until then, trachoma had been mainly spread among peoples of the Finnish race, who lived along the shores of the Volga. The Tartars founded three states in Russia: the Kingdoms of Kasan, Astrakhan, and Crimea. The Tartars of Kasan and Astrakhan are even today severely afflicted with trachoma (up to 25 to 50 percent).

The Crimean Tartars are, on the other hand, nearly free from this disease. This circumstance has aroused much wonder and interest in the investigators but has not, as yet, been accounted for. Dr. Fish considers that the mountain climate of Crimea makes the disease more mild. Yet, many people who live in mountainous regions (Caucasus, Altai, Algeria, Mexico) are afflicted by trachoma. There must be other reasons for this decrease in infection. One of them lies in intermarriage with healthy women.

It is known (Larousse) that the Crimean Tartars are not a pure race but a mixture of different peoples who had dwelt there before the Tartar conquest (Greeks, Goths, Italians). Moreover, the Tartars made frequent invasions into South Russia, where they captured women free from trachoma, while the Kasan and Astrakhan Tartars lost their independence in the days of John the Terrible, in the 15th century; that is, 200 years before the Crimean Tartars. They have lived since then in misery under Russian domination, and it is only natural that they married among themselves.

Now I wish to consider trachoma among a people until recently stateless and scattered all over the world, namely the Jews.

Some investigators (Krumbach, Herrenschward), inclined to "race" theories, consider the Jews as particularly subject to trachoma. Stanculeanu and Michael declare that the Jews, in their wanderings, propagate the disease throughout the world, the way the Arabs do. As a proof, they mention the fact that, in Amsterdam, Jews are infected with trachoma more than the Dutch population. Smith and Wibaut found in the schools, among 7,062 Jewish pupils, 623 who were suffering from trachoma (8.8 percent); whereas, among the other 66,418 pupils there were only 396 cases of the disease (0.59 percent).

In my opinion, the above-mentioned opinions are erroneous. All the statistics I could find prove unanimously that, all over the world, Jews suffer from the disease less than the surrounding population; wherever trachoma is widespread, the percentage of Jews afflicted with the disease will be comparatively high, yet always lower than that of the majority of the population. Following are a few illustrations.

In Lithuania, where Jews have been living for over 600 years, Rostkovsky found, in the Vilna district, 15 percent of the infected people among the Lithuanians and five percent among the Jews. In other districts the difference is even greater. Priaulgauskas stated that, in the schools of Chauliai, there was an incidence of 17 percent of the disease among thee Lithuanians and 2.3 percent among the Jews.

In Russia, among 170,458 recruits from all over the state, Lavrentiev found the lowest rate of trachoma among the Jews. Nearly the same figures were reported by Medvediev for South Russia (Ukraine). Among 220,000 people, the Germans showed an incidence of 16.9 percent; Czechs and Bulgars, 12.9 percent; Greeks, 10 percent; Jews, 4.9 percent; Moldavanians, 3.4 percent; Ukrainians, 2.45 percent.

In Western countries—England, France, Germany—where the rate of trachoma is, in general, low, it is as low among the Jews as other nationalities. In Palestine, according to Schimkin and Ticho, the rate of the disease in 1926 was about 15 percent among the Jews and over 40 percent among the Arabs. I have no exact information about America, but I believe that the picture is about the same as in Europe.

What are the reasons for such a high rate of trachoma among the Amsterdam Jews? The answer is to be found in the history of the Jewish settlement in this city. This settlement was started at the end of the 15th and the beginning of the 16th century, by Jewish refugees from Spain and Portugal.

As is well known, Spain was conquered in 711 A.D. by Arabs, who ruled it for over 700 years. No wonder trachoma spread among the Spaniards and the Spanish Jews who, having come from the Near East and North Africa, were probably, to a certain degree, infected with trachoma.

According to Graetz, 25,000 refugees fled to Holland, and many of them settled in Amsterdam. There they established a separate Jewish community in a quarter of their own. Under these conditions, the disease they had brought with them could spread among the Jews, from parents to children, but not among the Christians, who lived separately.

The second wave of Jewish immigration to Holland took place in the middle of the 17th century, when thousands of Jews fled from Ukraine and Poland after the rebellion and the massacres of Khmelnitzky. Part of them reached Amsterdam. It is natural that the refugees from Ukraine and Lithuania were more infected with trachoma than the Dutch.

The third wave reached Amsterdam at the end of the 19th century, after the pogroms of 1881 and after many Jews had been expelled from all the villages and many cities in Russia. These immigrants were headed for America, but part of them remained in the ports of Rotterdam and Amsterdam, many of them because they suffered from trachoma. All these facts account for the higher trachoma incidence among Jews in Amsterdam.

Two more instances of the influence of wars on the spread of the disease may be cited. Neither is sufficiently reviewed in the literature. The first occurred in Greece.

Greece, as a whole, is afflicted with trachoma; the incidence, however, is different in various regions. The highest rate of the disease is to be found in Peloponnesus (10.9 to 19 percent); in the Island of Zante, 27 to 28 percent; in Attica and Boeotia, 20.58 percent. A milder rate of infection is found in Macedonia: Saloniki district, 7.55 percent; Kozani, 10.4 percent; Droama, 7.41 percent; Thessalia, 3.96 to 5.32 percent. The lowest rate is found in Epire, 1.08 to 1.67 percent; in Florina, 0.85 percent; in Seres, 2.85 percent. How can such variety be accounted for?

According to Dascalopulos, the high rate of infection in Peloponnesus and Zanta Island is due to the Independence War (1821-1827). In these years the Egyptian troops of Ibrahim Pasha got hold of Peloponnesus, and contamination by trachoma followed. Part of the population fled to Zanta Island, then in the hands of the English where the population was very crowded and the disease spread widely.

The high rate of infection in Boeotia and Attica and other places is due to the war between Greece and Turkey in 1922. According to the Lausanne treaty, an interchange of population between Greece and Turkey was decided upon. The Anatolian Greeks settled in Greece and the Greek Turks in Anatolia. In all the places where refugees had settled, the rate of trachoma went up, especially in Attica and Boeotia, where the population was most crowded, and next to it, in Macedonia. Elsewhere, where no settlement had taken place, the rate of trachoma did not change.

Now consider the consequences of the first World War (1914-1918) and the Civil War in Russia (1917-1920).

The first World War had little effect on the propagation of trachoma, despite the fact that tens of millions of men were involved in it. In Germany, England, and Italy, no propagation of trachoma was observed either during or after the war. In France, an increase of trachoma was reported, which may be explained by the presence of colonial troops. Elschnig found an increase of the

disease in Czechoslovakia, owing to troops that had been staying in infected countries, such as Bosnia, the Volga districts in Russia, and in Siberia.

In Russia no propagation of infectious diseases in general and of trachoma in particular was reported. The Civil War in Russia, however, with its sequels, brought about a flare-up of infectious diseases and, among them, of trachoma. The destruction of public and state institutions, famine in a great part of Russia, the lack of necessities, a great insecurity as to the future—all of these brought about a complete neglect of the most elementary laws of hygiene. Millions of displaced people, who had left their homes either directly because of the Civil War or in quest of bread, contributed to the propagation of infectious diseases. As an illustration take the earlier and present statistics of the department of Voroniej. According to Ginsbourg (1901), among 5,804 country school pupils, there were only 215 cases of trachoma (3.6 percent); among recruits, two percent. Ginsbourg (1901) reported the rate of the disease in the department of Voroniej as three percent. Maslennikoff organized a general inspection of 20 villages, possessing 49,774 inhabitants; 34,189 of them were examined and 1,639 cases of trachoma (4.8 percent) were found; 5.45 percent among men; 6.64 percent among women; 2.51 percent among children below 15 years of age; 10.62 percent among school pupils.

On comparing the two sets of statistics, we observe an increase of the disease among school pupils from 3.6 percent to 10.62 percent; among adults from three percent to 4.8 percent. The low rate among children below 15 years of age proves that, in the department of Voroniej, no contagion from parents to children took place, as in the case of endemic trachoma; adults had been infected by adults who had passed there during the period of Civil War and famine. This is corroborated by the fact that recent trachoma among adults was found much more frequently than under normal conditions.

Summary

An endeavor is made to answer the question why trachoma is unequally distributed in different countries and among different nations.

It is my opinion that trachoma originated in one or a few places among certain tribes, whence it spread throughout the world following the migrations of those tribes. The nations in which the disease did not originate remained trachoma-free, or were infected later on as they came into contact with trachoma-contaminated nations.

In this process, the main part was played by historic events, such as mass migrations, wars, and foreign invasions.

I am of the opinion that Central Asia is one of the centers in which trachoma originated, considering that all the peoples coming from there have been infected with the disease. Mongolian and Ugro-Finnish tribes, peopling nearly the whole of Asia and a considerable part of Europe, are even now afflicted with a high incidence of trachoma.

The other region where trachoma probably originated is Arabia. The Arabs propagated the disease in all the countries they conquered—South Asia, North Africa, and Spain.

The role of two peoples—Lithuanians and Jews—in the propagation of trachoma, has been described. Lithuania is the center from which trachoma spread among the neighboring countries and peoples—Germans, Poles, Russians, and Jews.

As for the Jews, it is felt that they cannot be considered as propagators of trachoma, since the incidence of their infection is lower than that of of the surrounding population. The only exception is in the city of Amsterdam where the rate of trachoma among the Jews is much higher than among the Dutch population. There is a historic reason for this, namely, the many waves of Jewish immigration to Holland, the immigrants being highly contaminated by trachoma, either Spanish-Arabian or Russian-Ukranian in origin.

The influence of wars on the spread of trachoma is illustrated by the Napoleonic campaigns, the Greco-Turkish wars, and World War I, and these examples could be duplicated many times.

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COMPLICATIONS OF CATARACT SURGERY*

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During the years 1946 to 1950 inclusive we performed cataract extraction upon 565 eyes of private patients. In this series all types of cataract except congenital and traumatic were included. Intracapsular extraction was attempted in all except 20 instances in which there were definite indications in favor of the extracapsular operation. The capsule ruptured in 77 cases and was withdrawn separately and completely in 30.

Our method of choice is phakoerisis with continuous suction being supplied by a small Jordon electric pump, as recommended by Castroviejo. Having become accustomed to this method more than 10 years ago we still prefer it to that using the Bell erisophake or its later modification by Harrington. In only a few cases in this series was the forceps method used.

Preoperative preparation consists of nembutal (1.5 gr.), atropine (one percent) in the eye, and a saline enema the night before operation. One hour before operation the patient is given atropine (one percent) in the eye, and sodium amytal (3.0 gr.), or demerol (50 to 100 mg.), the latter in more apprehensive patients.

In the operating room cocaine hydrochloride (four percent) is instilled in the eye every two minutes for four doses. Akinesia of the lids is obtained by injecting four-percent novocain containing adrenalin (five drops) to the ounce, according to the technique of Van Lint, and a retrobulbar injection into the muscle cone is made with 1.25 to 1.5 cc. of the same novocain solution. A

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† By invitation.

solution of four-percent cocaine (10 drops), adrenalin (10 drops), and four-percent novocain (20 drops) is injected beneath the superior rectus, along the other rectus muscles if the patient cannot hold the eye still, and also into the skin along the central lid margins.

Lid sutures of 4-0 silk are used instead of a speculum and a similar suture is placed beneath the superior rectus to hold the eye in convenient position while the conjunctival flap is being prepared and the sclerocorneal sutures inserted. This is released before the eye is opened.

We prefer the use of preplaced sutures, and in approximately the first 100 cases of this series two scleroconjunctival sutures of 6-0 silk were used. In the second 100 we used two McLean sclerocorneal sutures of 6-0 silk; for the third and fourth 100 cases two McLean sutures of 4-0 plain gut, and in the remaining cases McLean sutures with 6-0 chromic gut have been used, two such sutures being placed in cases of complete iridectomy and three sutures in those with peripheral iridotomy and round pupil extraction.

For many years we have approached the removal of silk sutures with fear and trembling. To us it was always a formidable and occasionally a dangerous procedure. Therefore we have long looked forward to the time when a satisfactory absorbable suture material would be perfected. This was our reason for using 4-0 plain gut to which there are many well-known objections. In spite of these we preferred to use it instead of silk. The 6-0 chromic gut now available is fairly satisfactory. While it is not pliable and cannot be laid aside easily as can silk, we prefer to tolerate this difficulty and to be more painstaking in getting the sutures out of the way of our instruments, rather than to use silk and be forced to remove the sutures after operation. We have not found 6-0 chromic gut more irritating to the eyes than silk and have not seen it remained unabsorbed longer than twenty days.

We are not wholehearted advocates of round pupil extraction, for which we believe, as will be shown later, we have good reasons. In very old patients whose palpebral fissures are narrow and whose upper lid will cover the coloboma, and in those with rigid pupils which will not dilate widely, a complete narrow iridectomy is done.

A small incision is made with a keratome and enlarged with scissors. The cornea is held up by the conjunctival flap if two sutures have been placed, or by the central of three sutures by the assistant while the iridectomy or iridotomy is done.

The erisophake is inserted from the temporal side beneath the elevated cornea and placed below the center of the lens underneath the lower border of the pupil. It is held with light pressure against the lens for several seconds to allow the capsule to become adapted into the recesses of the suction cup. A combination of traction and pressure is used to rupture the lower zonule and the lens is delivered by tumbling.

If the erisophake releases the capsule before the zonule breaks it is replaced, but if the release occurs after rupture of the zonule the erisophake is not replaced. The lens is then delivered by the Smith-Indian technique, with expression hook and spoon.

The conjunctiva is closed with interrupted 4-0 plain gut. Often at the completion of the suturing of the conjuctiva the anterior chamber has begun to reform. If it has not, a bubble of air is injected into the chamber.

Eserine ointment (one percent) is instilled in all eyes except those with complicated cataracts. Atropine is used in these. A dressing with protective shield is placed over the operated eye, the other being left uncovered. The patient is allowed to turn to the unoperated side. The head of his bed may be elevated. Codeine and aspirin are administered for pain, and demerol if codeine is contraindicated. The dressing is changed daily and the patient allowed to be out of bed on the second day.

In this series 127 eyes had some sort of operative or postoperative complication

TABLE 1

COMPLICATIONS ENCOUNTERED*

1. Vitreous loss	37
2. Rupture of capsule	21
3. Delayed reformation of A.C.	15 (1)
4. Hemorrhage	14
5. Uveitis	33 (8)
6. Prolapse of iris	9 (5)
7. Glaucoma	33 (17)
8. Keratitis	11
9. Vitreous contact	5
10. Optic neuritis	4
11. Epithelization of A.C.	2
12. Sympathetic ophthalmitis	3
13. Subchoroidal hemorrhage	2
14. Phaco-anaphylaxis	1 (2)
15. Separation of retina	12 (6)
16. Rupture of hyaloid	2
17. Secondary membrane	16 (16)

* Figures in parentheses are the number of operations for the complication.

which either prevented or delayed recovery of good vision. Among these 127 eyes, complications diagnosed preoperatively were as follows:

Fluid vitreous	4
Subluxation of the lens	3
Complicated cataract	11
Glaucoma	5
Endothelial dystrophy	4
Diabetic cataract	2

The complications observed are classified in 17 categories as shown in Table 1. The total number of complications suffered by these 127 eyes was 220, therefore a considerable number of eyes had more than one complication. The relation of multiple complications is shown in Table 3.

VITREOUS LOSS (37 cases)

Table 2 is a study of the cases in which vitreous was lost, revealing that the loss occurred before delivery of the lens in 10, during delivery in 15, and after delivery in 12 instances.

In some eyes such as Cases 3, 17, 22, 24, and 36, when the vitreous is fluid or is present in the anterior chamber, it is almost inevitable that vitreous will be lost in spite of the utmost precautions to prevent it. However, in other cases something happens to increase pressure upon the vitreous which should not happen and which might be prevented. For instance, the patient, Case 15, who raised his head, or Case 31, who squeezed his lids, had not received enough sedation or akinesia, and we, as surgeons, should have been alert enough to recognize the situation and take steps to correct it before the accident happened. In the other three cases pressure upon the vitreous came from some source which we did not recognize, but which closer observation might have brought to light.

When vitreous loss occurs during delivery of the lens it is obvious that undue pressure is exerted upon the vitreous by some faulty maneuver of the surgeon. This is true also when the loss occurs after delivery. Such steps as drawing up the sutures, removing a ruptured capsule, irrigating cortical remnants or reposition of the iris may easily cause vitreous loss and should be performed with the utmost delicacy possible in order to avoid injury to the hyaloid membrane. Probably we become complacent and fail to exercise the caution we should in every case. This idea is supported by evidence of several other cases in this series—nine highly myopic eyes, 12 or more diopters, and five complicated cataracts which we had watched develop after chorioretinitis. These cases, in which we knew there was abnormal vitreous, were operated upon without vitreous loss or any other complication. Perhaps our psychologic attitude was such that we operated with more care and caution. At least that is the indictment we have placed upon ourselves and we have resolved to operate upon every case as though we knew it to have fluid vitreous. By so doing we hope to reduce the incidence of vitreous loss.

RUPTURE OF CAPSULE (21 cases)

We have noticed in phakoerisis that occasionally the posterior capsule will strip off and remain unnoticed unless the extracted lens is examined minutely. Several times we thought we had accomplished complete intra-

TABLE 2

VITREOUS LOSS

Case No.	Vitreous	Condition	Vision
		Before Delivery	
3	Fluid	No iridectomy. Loop. Up-drawn pupil. Iridotomy	20/70
15	Normal	Patient raised head, expelled vitreous and lens. Uveitis	20/100
17	Watery	Complicated Cataract. Collapse of globe. Uveitis	0
18	Normal	Before zonule ruptured	20/25
22	Fluid	As incision completed expulsive gush of vitreous and lens	20/30
24	Fluid	Subluxation. Vitreous in anterior chamber. Collapse	20/30
29	Normal	Nasal presentation replaced. Loss temporally as cut suture re-inserted	20/20
31	Normal	Chronic glaucoma. Poor akinesia. Squeezed lids. Glaucoma	20/50
34	Normal	Before zonule ruptured	20/20
36	Normal	Subluxation. Vitreous in anterior chamber. While enlarging incision	20/30
		During Delivery	
5	Normal	Small amount. Separated retina	0
6	Normal	Small amount. Separated retina	0
7	Normal	Small amount. Separated retina	0
8	Normal	Small amount. Glaucoma	20/50
12	Normal	Small amount. Hemorrhage	20/30
16	Normal	Small amount	20/30
19	Normal	Small amount	20/40
21	Fluid	Moderate amount. Superior rectus suture not released. Separated Retina	0
23	Fluid	Moderate amount	20/25
25	Normal	Small amount. As zonule ruptured	20/30
26	Normal	Small amount	20/25
28	Fluid	Moderate amount. Continued while tying sutures	20/60
30	Normal	Small amount. Glaucoma	20/20
35	Normal	Small amount	20/20
37	Normal	Small amount	20/30
		After Delivery	
1	Fluid	On reposition of iris. Uveitis. Glaucoma	20/40
2	Normal	After wound was closed. Uveitis	10/20
4	Normal	R.C. during air injection. Discission	20/30
9	Normal	While drawing up sutures. Uveitis. Glaucoma	20/80
10	Normal	In reposition of Iris. Hemorrhage. Uveitis. Prolapse of iris. Glaucoma	20/40
11	Fluid	While placing conjunctival sutures	H.M.
13	Normal		20/30
14	Normal	R.C. during irrigation. Discission	H.M.
20	Normal	R.C. while trying to remove capsule. Hemorrhage. Keratitis	5/200
27	Normal	R.C. during irrigation	20/20
32	Normal	Chronic Glaucoma	20/40
33	Normal		20/20

capsular extraction only to find later that a secondary membrane had developed.

While this complication usually is not of too serious import, it can be the cause of others more grave, such as vitreous loss while trying to remove the capsule or cortical remnants. Discission of a capsular membrane becomes necessary in a rather large percentage of cases and this operation is not without danger. For instance, Case 68 developed uveitis and optic neuritis after discission and suffered total loss of vision.

DELAYED REFORMATION OF ANTERIOR CHAMBER (15 cases)

Only three of these patients, Cases 39, 44, and 48, had actually a delayed closure of the wound. One, Case 48, required operation to resuture the wound. The others suffered loss of the anterior chamber for variable lengths of time, none longer than five days, during their hospital stay. The seriousness of this complication is shown by the fact that seven of these eyes developed glaucoma, three developed uveitis, and in one this com-

TABLE 3

RELATION OF MULTIPLE COMPLICATIONS

Case No.	V.L.*	R.C.	D.R.	H	U	P.I.	G	K	V.C.	O.N.	E.P.	S.O.	C.H.	P.A.	S.R.	R.H.	S.M.
1	+				+		+†										20/40
2	+				+												†10/200
4	+	+															+†20/30
5	+														+†		0
6	+														+		0
7	+														+†		0
8	+						+										20/50
9	+				+†		+										20/200
10	+			+	+	+†	+†										20/40
12	+			+													20/30
15	+				+												20/100
17	+				+												0
20	+			+				+									5/20
21	+														+		0
30	+						+†										20/20
31	+						+										20/50
38			+				+		+								20/70
39			+		+		+†					+					0
40			+		+	+†	+					+					20/30
41			+				+†										20/30
42			+		+		+†										20/50
43			+		+†				+								0
44			+				+										0
48		+	+†														20/50
51			+				+	+									20/25
52				+				+	+								0
55				+				+									†20/50
56	+	+		+													+†20/80
58				+	+†	+	+†										†20/200
60		+		+													+†H.M
61							+									+	20/30
64				+			+										20/20
66		+			+†		+†										0
68		+			+					+							+† 0
69					+					+							20/70
72					+		+†									+	20/25
73					+	+											20/40
74					+					+							†20/50
75		+			+												+†20/50
76					+										+		20/200
77					+		+†										0
82					+			+									0
83					+		+										20/200
84		+			+												H.M.
85					+		+										0
89					+										+		0
91					+	+						+					0
92						+	+										20/25
93					+	+						+					0
98							+	+									20/50
100					+						+						0
101							+†	+	+								20/200
107							+†				+						20/80
111		+												+			20/100
114	+														+†		0
117		+															+†20/20
118		+															+†20/25
119		+															+†20/30
120		+															+†20/15
121		+															+†20/20
122		+															+†20/40
123		+															+†20/20
124		+															+†20/20
125		+															+†20/20
126		+															+†20/20
127		+															+†20/40

* Classifications refer to categories in Table 1.
† Operation after the particular complication.

plication was the first link in the chain of events which led to sympathetic ophthalmitis.

The use of two or three sclerocorneal sutures greatly reduces the occurrence of delayed reformation of the anterior chamber.

HEMORRHAGE (14 cases)

Included are only those eyes in which the hemorrhage invaded the vitreous or occurred only in the vitreous, and played a definite part in preventing or delaying recovery of good vision. More than half of these eyes developed other complications as will be shown later. In addition there were 49 eyes which had hyphema, sometimes filling the anterior chamber, but which did not delay convalescence. In Case 57 the vitreous was so filled with blood that she had no fundus reflex for 10 months, but her vision now is 20/70 and

is still improving slowly as the vitreous clears.

In 100 consecutive cases we used vitamin K (Synkamin) preoperatively and daily for one week postoperatively, in an effort to reduce the occurrence of hyphema, but no reduction occurred.

Uveitis (33 cases)

Uveitis is one of the more frequent complications seen after cataract surgery, also one of the most serious. Nine of these 33 eyes lost all vision, not necessarily because of uveitis alone, but because the uveitis was an important part of the deteriorative process which led to destruction of vision. It will be shown later that 25 of these eyes had other complications. It was stated before that we are not advocates of the round pupil in all cases. The principal reason is that eight of these cases of uveitis had round pupil extractions. The pupil became occluded or secluded in four and it was necessary to do an iridectomy or iridotomy. Two cases developed glaucoma and one lost all vision. Perhaps we should not have tried round pupil extraction in these eyes; at that time we were less careful in determining the mobility of the pupil. The trauma of stretching the sphincter to deliver the lens probably was a major factor in causing the uveitis.

Since cortisone has been available it has been of great help in the treatment of this complication. This medication is administered by topical instillation in anterior uveitis and parenterally or by mouth in cases with posterior involvement.

Prolapse of iris (9 cases)

Because three of the eyes which had prolapse of iris went on to develop sympathetic ophthalmitis, we look upon this complication as one which may have dire consequences and should therefore be treated most carefully. If the prolapse is judged to require surgical correction we would recommend dissection and excision of the prolapsed iris and resuturing the wound rather than destroying it by actual cautery, because two of our cases of sympathetic ophthalmitis followed cauterization of the prolapse. Two others were corrected by dissection and excision, but did not require resuturing of the cornea, while four did not require surgery. Two of these consist only of a tiny prolapse surrounded by a filtering cicatrix and have caused no apparent symptoms. We have not felt it advisable to disturb them.

Glaucoma (33 cases)

In nearly every instance glaucoma developed as a result of, or at least concurrent with, some other complication. Seventeen of these eyes required surgery. Cyclodialysis was done in all of them. In 22 cases the glaucoma was controlled. Nine did not require the use of miotics. It is of interest that the patient who had glaucoma capsulare did very poorly. Her tension was never controlled by any miotic or operation. Her vision, good at first, deteriorated rapidly and the disc showed deep cupping and optic atrophy.

Keratitis (11 cases)

Four of these eyes had endothelial dystrophy before operation. This progressed rather rapidly after operation, involving stroma and epithelium and causing dense cloudiness of the cornea. Three eyes had severe intraocular hemorrhage; two had prolonged contact between the vitreous and cornea; one was due certainly to injury at operation; while the others were associated with persistent striate keratitis which may or may not have been due to injury of the corneal endothelium.

Vitreous contact (5 cases)

In two of these eyes the contact persisted long enough to produce clouding of the cornea. Two others had long contact without corneal clouding. In the remaining eye the vitreous remained in contact with the cornea for some time after delayed reformation of the anterior chamber and probably was a factor in causing glaucoma.

Optic neuritis (4 cases)

We have not seen this condition listed elsewhere as a complication of cataract surgery. In Case 68 the posterior capsule stripped off at operation. Vision was good for four months when it became defective because of thickness of the capsular membrane. Immediately after discission a mild uveitis occurred with slight congestion and haziness of the vitreous. The optic disc became very much swollen, three or four diopters, and vision failed rapidly. At the termination of the active process there was a gray atrophy of the nerve. Case 105 developed optic neuritis without any other complication and has lost all useful vision. Case 74 had moderately severe uveitis and optic neuritis which subsided leaving her with 20/50 vision. Case 69 had uveitis following operation which persisted, although vision was 20/30 during this time. Fifteen months after operation optic neuritis developed and still is present after more than two years. It has failed to respond to any treatment. The vitreous is still cloudy and the optic disc swollen about three diopters; vision is 20/70.

Epithelization of anterior chamber (2 cases)

For four months after operation, Case 100 had 20/20 vision and no trouble. The eye then became painful. He was treated in another city where two paracenteses were done for glaucoma. When we saw him six months after operation, vision was 20/30 and the tension was normal. We made a diagnosis then of epithelial downgrowth over the iris and posterior corneal surface. Vision deteriorated rapidly, and epithelization progressed steadily during the next year but the tension was soft and the eye painless. When glaucoma developed again and the eye became painful he consented to enucleation. The diagnosis was confirmed by pathologic examination.

Case 106 had chronic wide-angle glaucoma before operation. The tension remained elevated after the cataract was removed. We made a tentative diagnosis of epithelization because of a faint gray line which seemed to be progressing downward on the posterior surface of the cornea. There also was some vascularization on the anterior corneal surface. Beta radiation therapy was administered with a total dosage of 920 r. Progress of the gray line stopped thereafter and the vascularization cleared. Although we cannot be certain of the diagnosis we believe this case belongs in this category.

Sympathetic ophthalmitis (3 cases)

Case 39. Intracapsular extraction, right eye, February, 1946, was followed by delayed reformation of the anterior chamber and glaucoma, which was relieved by cyclodialysis in March, 1947. Vision was 20/20−; tension 15 mm. Hg (Schiøtz).

Case 40. Intracapsular extraction, left eye, September, 1947, was followed by prolapse of iris which was cauterized with actual cautery. Vision 20/30+, no congestion, media clear, fundus normal, tension 20 mm. Hg (Schiøtz). Left eye 20/30+, no congestion, media clear, fundus normal, tension 20 mm. Hg (Schiøtz). December 26, 1947, right eye 4/200, anterior chamber flat, vitreous cloudy, no fundus details, tension 12 mm. Hg. Left eye 20/50, anterior chamber shallow, vitreous hazy, fundus normal, tension 12 mm. Hg. During the next three months vision was reduced to 10/200 or less, both eyes were mushy soft, and showed corneal precipitates and pigment on the face of the vitreous. Dense posterior synechias developed in the right eye. Tension in this eye rose slowly until it was again glaucomatous and vision failed completely. The left eye has maintained constantly low tension and 11 months after the ophthalmitis began vision improved until vision was 20/30 where it has remained for more than two years. Both eyes show widespread retinal degeneration with pigmentation. We presume that the right was the inciting eye as it was the first to show inflammatory signs, although only a few days before the

left eye also became involved. No treatment had any apparent effect upon the course of the disease.

Case 91. Intracapsular extraction with round pupil June 13, 1946. At first dressing there was a large prolapse of iris. Immediately the conjunctival flap was raised and the prolapse destroyed by actual cautery. The wound edges were coapted nicely and no sutures were placed. The eye did not respond well, remaining irritable and painful. Although the prolapse did not recur the wound was slow to heal soundly and was cauterized several times with trichloracetic acid. Vision was 20/70. July 29, 1946, the left eye, upon which we had done intracapsular extraction in 1942, suffered occlusion of the central retinal vein reducing vision from 20/20 to 20/150. August 27, 1946, the right eye showed some exudate in the anterior chamber and tension was subnormal. September 13, 1946, both eyes showed ciliary congestion, there was more exudate in the right eye, and the site of the prolapse was slightly invaginated. The right eye was enucleated September 14, 1946. The left eye did not recover but progressed slowly to phthisis bulbi.

Case 93. A 43-year-old man had posterior capsular and subcapsular cataract, bilateral, which made us suspect complicated cataract, although there was no history of other eye trouble. January 10, 1949, intracapsular extraction of right eye with round pupil. A tiny prolapse, completely covered with conjunctiva occurred at the 12-o'clock position. For two and one-half months there was no trouble. Vision was 20/20. March 11, 1949, iridocyclitis with cloudy vitreous, vision 20/100. The iridocyclitis subsided in 10 days and the vitreous cleared so that by May 5, 1949, vision again was 20/20−. June 16, 1949, vitreous cloudy, vision 20/70. Vision became worse until on September 22, 1949, in the right eye it was 20/400, and the left eye showed signs of beginning iridocyclitis.

The patient was told that we suspected sympathetic ophthalmitis but he refused enucleation. Activity of the inflammation in the right eye subsided slowly during the next four months. By January 20, 1950, the eye was quiet and vision 20/50, where it remained until March 7, 1950, when another relapse occurred. The vitreous became very cloudy and vision fell to 20/200. March 21, 1950, complete basal iridectomy was done.

Following operation the right eye deteriorated rapidly, the iris became vascularized and degenerated, the anterior chamber shallow, and tension rose to 40. The eye finally was enucleated July 21, 1950.

Meanwhile the left eye had developed corneal precipitates and posterior synechias, but at no time showed more than mild congestion. Since enucleation of the right eye it has improved steadily, until at present it has 20/50 vision in spite of well-advanced cataract.

All three of these patients were treated vigorously, the last case receiving cortisone both topically and parenterally, also tuberculin therapy and streptomycin and promizole, as he had a positive skin test from the beginning. Nothing appeared to be of real benefit. If it had been given earlier, cortisone might have been effective in the last case. We believe it helped considerably in clearing the left eye.

Subchoroidal hemorrhage (2 cases)

One of these occurred on the afternoon of the day of operation when the patient developed severe nausea and vomiting. Her gall bladder had been removed and she was subject to nausea and vomiting, but had not told us about this tendency before operation. Had we known about it we might have been able to forestall the attack which caused the hemorrhage.

The second case suffered hemorrhage during sleep three days after operation. She had far advanced generalized arteriosclerosis and mild hypertension.

Phaco-anaphylaxis (1 case)

This patient had an immature cataract and

immediately after operation developed a severe reaction to lens protein. Later the eye was opened, the retained cortex removed and a wide iridectomy done. Thereafter she became sensitive to all mydriatics and the pupil was occluded. Later when all reaction subsided an iridotomy was done, giving a clear pupil area. Vision then was 20/50. She has since had the other eye operated upon elsewhere with excellent result, so that she does not use the first eye and its vision has dropped to 20/100.

Separated retina (12 cases)

Total separation of the retina was noticed immediately after operation in two cases. Preoperative findings led us to believe that they probably existed beforehand. In four cases the separation occurred within six months after removal of cataract, while in the others it occurred between one and three years after removal.

Six eyes were operated upon; two of them had two operations. In only two eyes was the retina replaced. Vision in these is 20/50 and 20/20. The latter eye required a second operation two months after the first.

Rupture of the hyaloid (2 cases)

In both of these cases the rupture occurred several months after operation and was accompanied by glaucoma which could not be controlled without surgery. Both have had cyclodialysis after which the tension has remained low.

Secondary membrane (16 cases)

In these cases discission was necessary. With the exception of Case 68, already mentioned, no mishap occurred.

Visual results

The final visual acuity, or the latest recorded vision, in these 127 eyes indicates that 47 have less than 20/200. It is certain that 35 of these lost their vision as a direct result of complications of cataract surgery, while 12 had other ocular diseases which contributed to the loss of vision.

TABLE 4

Visual Results

Totally blind	28
Less than 20/200	11
20/200	8
20/100	4
20/80	3
20/70	5
20/60	2
20/50	11
20/40	10
20/30	18
20/25	8
20/20	19

The presence in 68 eyes of vision good enough to enable them to read attests to the fact that the eyes can sometimes suffer an amazing amount of disease and still recover useful function.

Comment

Review of the complications occurring in his surgical practice will be enlightening and beneficial to any surgeon. It will point out his mistakes in judgment as well as technique. By application of knowledge thus gained he will be able to improve both and thereby reduce the occurrence of complications.

332 West Broadway (2).

THE IMPORTANCE OF CORRECTED CURVES IN SPECTACLE LENSES*

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For many years the advertisements have proclaimed the advantages of corrected curves for spectacle lenses. Competition provokes great effort to indicate the superiority of a certain brand so that refractionists will specify the brand even in lower powers.

Both ophthalmologists and opticians can be misled. The former may lack basic knowledge of optics, and the latter may believe the eye to be a fixed structure with rigid optical characteristics. It is most certain that no human eye maintains under various visual tasks and lighting conditions a focal length to the precision of one one-thousandth of a millimeter as suggested by the schematic eye.

Some advertising is obviously outside the knowledge or control of lens designers. In at least one instance, it has erroneously suggested that corrected curves can reduce all aberrations; increase the depth of focus; correct coma; reduce chromatism, barrel and pincushion distortion, and curvature of image. Even if this were possible, it would be illogical, because such errors exist in the normal eye without corrective lenses, and apparently do not interfere with visual efficiency.

For routine refractive purposes, only three types of aberration are reduced, and can be called important. One is chromatism in bifocal segments which will not be further discussed. The others are astigmatism and spheric power changes in vision through the spectacle lens margins. These two cannot be totally eliminated in the same lens, although either can be corrected at some expense of the other.

In order to evaluate the importance of corrected curves in spectacle lenses, the whole theory should be reviewed in a simple and unprejudiced manner for ophthalmologists. Such a review does not occur in medical literature. One paper written for physicists is not available.[1] Trade literature is inadequate and the patents on corrected curves are purposely confusing and nonrevealing.

William H. Wollaston,[2] (1803) found by mathematics that ". . . the more nearly any spectacle glass can be made to surround the eye, in the manner of a globular surface, the more nearly will every part of it be at right angles to the line of sight, the more uniform will be the power of its different parts, and the more completely will the indistinctness of lateral objects be avoided." Although his lenses are still too deep curved to be practical, they did correct marginal astigmatism. For certain purposes outside the domain of this discussion, they may be superior.

The principle of correction of ametropia by spectacle lenses is illustrated in Figure 1. For the myopic eye "M," the "far point" at which an object begins to blur without a corrective lens is found to be 50 cm. in a minus 2.00 diopter myope, "B"; and 100 cm. in a 1.00 diopter myope, "A." Just below "M" in the principal plane "P" is a minus lens which makes rays coming from a distance appear to have come from the same distance as "B," or "A."

When such a lens is superimposed on the myopic eye, objects at a distance are in focus on the retina. Without the lens, the retinal image is in conjugate focus with "B" or "A." With the lens, the retinal image is in conjugate focus with objects at a distance.

Since rays which come to focus on the retina of a hypermetropic eye "H" must be converging, without accommodation or corrective lens, no object in space can possibly be conjugate. The theoretic condition for conjugate foci would be as illustrated in the lower part of Figure 1.

* From the Department of Ophthalmology and the Oscar Johnson Institute of the Washington University School of Medicine.

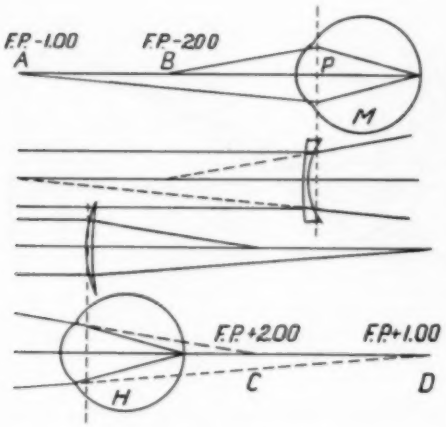

Fig. 1 (Miles). The "far point" of an uncorrected myopic eye is one meter away in a −1.0D. myope "A," and one-half meter away in a −2.0D. myope "B." If the eye is hypermetropic "H," no object in real space can focus on the retina, so the "far point" is represented to extend from the converging rays behind the eye to "C" and "D." In each case, the spectacle lens which has the appropriate focal length equal to the distance of the "far point" from the principal plane corrects the ametropia.

The "far point" is determined by extending the original converging rays along the broken lines to an imaginary point behind the eye. In case of hypermetropia of two diopters, this point would be 50 cm. behind the principal plane of the eye. In hypermetropia of one diopter, this point would be 100 cm. behind. These points are labelled "C" and "D" in Figure 1.

Just above the hypermetropic eye "H" is the correcting lens which would correspond perfectly in focal length with the distance between the principal plane and the "far point" of the eye, "C" or "D."

Any uncorrected myopic or hypermetropic eye will have a "far point" the conjugate retinal image of which will be sharp. As the eye moves freely to look in all directions, the "far point" is carried along on the visual line, describing in space a partial sphere. This "far-point sphere" is illustrated in Figures 2 and 3 for the myopic and the hypermetropic eye. (Since a "far point" is always more remote in upward gaze than lower, the "far-point sphere" actually tilts.) The dioptric distance of the "far point" from the eye should be measured from the principal plane, not the rotation center.

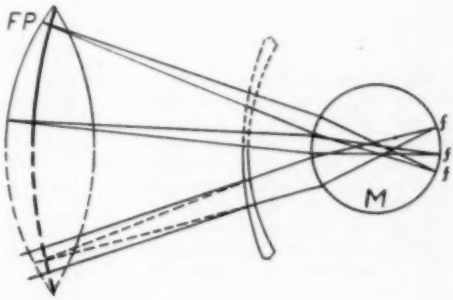

Fig. 2 (Miles). In all directions of gaze, the "far point" is fixed on the visual line describing the "far-point sphere." The correcting lens in myopia collects rays from far distance and makes them diverge as though they originated on the "far-point sphere."

In Figures 2 and 3 the rays bend at the eye surface, In Figure 2, an object anywhere on the sphere "FP" would focus on the retina at "f" without benefit of the correcting lens. Furthermore, parallel rays entering toward the eye rotation center are rendered divergent by the correcting lens regardless of their direction source. The divergence is such that when traced back as in the broken lines, the rays would appear to have come from a point on the "far-point sphere."

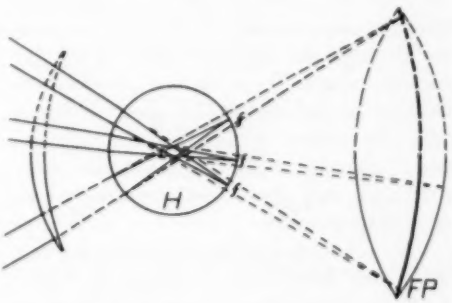

Fig. 3 (Miles). In hypermetropia, the plus lens will focus parallel rays from any direction so that disregarding the eye, they will focus on the "far-point sphere."

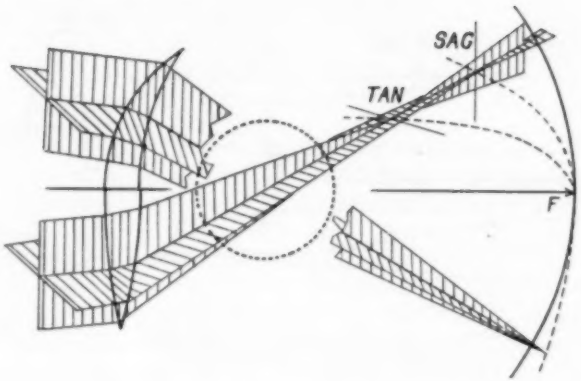

Fig. 4 (Miles). The image of a distant object on the "far-point sphere" (solid line) of a hypermetropic eye corrected by a meniscus lens of plus 8.0D. is a point, whether rays are selected through the optical center of the lens or through the margin. The corresponding image through a biconvex lens is astigmatic and too near the eye.

In Figure 3, imaginary objects anywhere on the "far-point sphere" would focus on the retina at "f" without benefit of lens if the corresponding converging rays could be introduced through the cornea. This is accomplished by the appropriate lens illustrated, which would make parallel rays from a distance converge so that disregarding the eye, the focus would fall on the "far-point sphere."

It is therefore axiomatic that any correcting ophthalmic lens must put the image from a distant object exactly on the point in real or imaginary space occupied by the far point of the eye. The object is optically moved from distance to the appropriate position in space, from which the ametropic eye can see it.

Since the spectacle lens remains fixed while the eye rotates behind it, the lens must have the same effective power for rays passing through any lens area toward the eye rotation center. To attain this coördination, the two curved surfaces of spectacle lenses must be chosen to produce the required power not only in the center but also in the periphery and also avoid the astigmatism of oblique pencils.

The importance of lens shape in the case of a plus 8.0D. lens is shown in Figure 4. Above the horizontal axis, the correcting lens is bent so that parallel rays in the horizontal and vertical planes arising from a distant-point object are refracted equally to fall on a point just beyond the "far-point sphere" of the eye (solid line). Similar rays parallel to the horizontal axis would fall on a point on the "far-point sphere" at "F." In order to eliminate marginal astigmatism, about 0.10 diopters of correcting lens power must be sacrificed.

Similar rays entering the biconvex plus 8.0D. lens misbehave. Rays in the horizontal plane converge too strongly due to their direction of incidence on the back surface

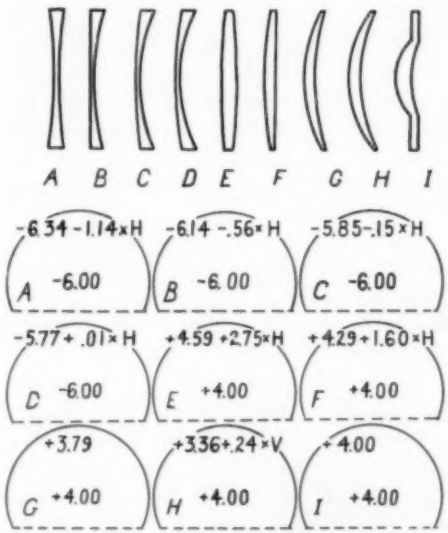

Fig. 5 (Miles). The effective power in 30-degrees upward gaze through spectacle lenses of different shapes in which the central vertex power is minus 6.0D. or plus 4.0D.

TABLE 1

SWAINE'S[3] CALCULATIONS (1938)

PART I

Diopters Front Surface	Effective power with gaze elevated 15° (Swaine)[3]				
22	−23.44 −2.18 ×H	−21.14 −2.48 ×H	−18.80 −1.91 ×H	−16.69 −1.32 ×H	−14.52 −1.07 ×H
20	−23.00 −2.57 ×H	−20.84 −1.92 ×H	−18.64 −1.24 ×H	−16.48 −1.04 ×H	−14.33 −0.73 ×H
18	−22.79 −1.94 ×H	−20.58 −1.42 ×H	−18.42 −1.02 ×H	−16.29 −0.70 ×H	−14.19 −0.43 ×H
16	−22.52 −1.41 ×H	−20.36 −1.00 ×H	−18.24 −0.67 ×H	−16.14 −0.42 ×H	−14.05 −0.22 ×H
14	−22.31 −0.96 ×H	−20.19 −0.63 ×H	−18.09 −0.38 ×H	−16.02 −0.18 ×H	−13.96 −0.04 ×H
12	−22.14 −0.72 ×H	−20.04 −0.37 ×H	−17.97 −0.14 ×H	−1 .92 −0.00	−13.89 +0.09 ×H
10	−22.01 −0.33 ×H	−19.94 −0.12 ×H	−17.89 +0.03 ×H	−1 .86 +0.13 ×H	−13.85 +0.20 ×H
8	−21.93 −0.12 ×H	−19.88 +0.04 ×H	−17.84 +0.14 ×H	−15.83 +0.20 ×H	−13.83 +0.22 ×H
6	−21.88 −0.02 ×H*	−19.85 +0.13 ×H	−17.83 +0.20 ×H	−15.83 +0.22 ×H	−13.84 +0.21 ×H
4	−21.88 +0.08 ×H	−19.85 +0.17 ×H	−17.85 +0.19 ×H	−15.85 +0.18 ×H	−13.88 +0.16 ×H
2	−21.92 +0.07 ×H	−19.91 +0.11 ×H	−17.91 +0.12 ×H	−15.92 +0.10 ×H	−13.94 +0.06 ×H
0	−22.01 −0.02 ×H	−20.00	−18.00 −0.01 ×H	−16.01 −0.05 ×H	−14.00 −0.09 ×H
−2	−22.13 −0.17 ×H	−20.13 −0.18 ×H	−18.13 −0.20 ×H	−16.14 −0.24 ×H	−14.14 −0.20 ×H
	−22	−20	−18	−16	−14
	Central vertex power diopters				
	+5.50 = −21.88 +0.50 = −21.99	+8.50 = −19.89 +0.00 = −20.00	+10.25 = −17.91 + 0.25 = −17.99	+12.00 = −15.92 + 0.62 = −15.98	+13.37 = −13.94 + 1.25 = −13.96

PART II

22	−12.38 −0.74 ×H	−10.27 −0.46 ×H	−8.18 −0.25 ×H	−6.16 −0.18 ×H	−4.05 −0.09 ×H	−2.02 −0.03 ×H
20	−12.24 −0.48 ×H	−10.15 −0.30 ×H	−8.09 −0.17 ×H	−6.05 −0.08 ×H	−4.02 −0.04 ×H	−2.00 −0.01 ×H
18	−12.10 −0.27 ×H	−10.06 −0.14 ×H	−8.02 −0.05 ×H	−6.00	−3.99 +0.02 ×H	−1.99
16	−12.01 −0.09 ×H	− 9.98 −0.01 ×H	−7.95 −0.04 ×H	−5.96 +0.05 ×H	−3.95 +0.05 ×H	−1.98 +0.03 ×H
14	−11.94 +0.04 ×H	− 9.92 +0.09 ×H	−7.92 +0.10 ×H	−5.93 +0.09 ×H	−3.95 +0.06 ×H	−1.97 +0.03 ×H
12	+11.88 +0.14 ×H	− 9.88 +0.15 ×H	−7.90 +0.12 ×H	−5.92 +0.10 ×H	−3.94 +0.07 ×H	−1.97 +0.03 ×H
10	−11.85 +0.19 ×H	− 9.86 +0.18 ×H	−7.89 +0.15 ×H	−5.91 +0.10 ×H	−3.94 +0.06 ×H	−1.97 +0.02 ×H
8	−11.84 +0.21 ×H	− 9.87 +0.18 ×H	−7.89 +0.13 ×H	−5.92 +0.08 ×H	−3.95 +0.04 ×H	−1.98 +0.01 ×H
6	−11.86 +0.18 ×H	− 9.89 +0.14 ×H	−7.91 +0.07 ×H	−5.94 +0.04 ×H	−3.97	−1.99 −0.01 ×H
4	−11.90 +0.12 ×H	− 9.92 +0.07 ×H	−7.95 +0.02 ×H	−5.97 −0.02 ×H	−3.99 −0.05 ×H	−2.00 −0.04 ×H
2	−11.95 +0.01 ×H	− 9.98 −0.04 ×H	−8.00 −0.08 ×H	−6.02 −0.12 ×H	−4.03 −0.11 ×H	−2.02 −0.08 ×H
0	−12.05 −0.13 ×H	−10.06 −0.18 ×H	−8.07 −0.21 ×H	−6.07 −0.21 ×H	−4.07 −0.18 ×H	−2.04 −0.12 ×H
−2	−12.15 −0.33 ×H	−10.16 −0.35 ×H	−8.15 −0.36 ×H	−6.14 −0.34 ×H	−4.11 −0.27 ×H	−2.06 −0.16 ×H
	−12	−10	−8	−6	−4	−2
	+14.62 = −11.97 + 1.87 = −11.97	+15.87 = −9.96 + 2.62 = −9.96	+15.75 = −7.94 + 3.75 = −7.97	+18.00 = −6.00 + 4.62 = −5.98	+18.62 = −4.00 + 6.00 = −3.97	+18.00 = −1.99 + 7.00 = −1.99

PART III

22	+2.01 +0.02 ×H	+4.02 +0.02 ×H	+6.42 +0.03 ×H	+8.02 +0.05 ×H	+10.02 +0.09 ×H
20	+2.00	+3.99 −0.01 ×H	+5.98	+7.97 +0.02 ×H	+ 9.97 +0.06 ×H
18	+1.99 −0.02 ×H	+3.97 −0.03 ×H	+5.94 +0.01 ×H	+7.94 +0.07 ×H	+ 9.95 +0.06 ×H
16	+1.98 −0.02 ×H	+3.96 −0.03 ×H	+5.94 −0.02 ×H	+7.93 +0.02 ×H	+ 9.93 +0.10 ×H
14	+1.97 −0.02 ×H	+3.95 −0.02 ×H	+5.94	+7.93 +0.07 ×H	+ 9.94 +0.18 ×H
12	+1.98 −0.01 ×H	+3.96	+5.95 +0.05 ×H	+7.95 +0.14 ×H	+ 9.97 +0.28 ×H
10	+1.98	+3.97 +0.03 ×H	+5.97 +0.11 ×H	+7.98 +0.24 ×H	− 9.99 +0.44 ×H
8	+1.98 +0.02 ×H	+3.99 +0.08 ×H	+6.00 +0.19 ×H	+8.03 +0.36 ×H	+10.08 +0.59 ×H
6	+2.00 +0.05 ×H	+4.01 +0.15 ×H	+6.04 +0.29 ×H	+8.09 +0.51 ×H	+10.17 +0.80 ×H
4	+2.02 +0.09 ×H	+4.05 +0.22 ×H	+6.11 +0.42 ×H	+8.17 +0.69 ×H	+10.27 +1.04 ×H
2	+2.03 +0.13 ×H	+4.09 +0.31 ×H	+6.17 +0.57 ×H	+8.27 +0.90 ×H	+10.40 +1.31 ×H
0	+2.06 +0.17 ×H	+4.14 +0.41 ×H	+6.24 +0.73 ×H	+8.37 +1.12 ×H	+10.54 +1.62 ×H
−2	+2.09 +0.23 ×H	+4.20 +0.53 ×H	+6.33 +0.91 ×H	+8.50 +1.39 ×H	+10.70 +1.98 ×H
	+2	+4	+6	+8	+10
	+20.00 = +2.00 +10.00 = +1.98	+20.25 = +4.00 +12.00 = +3.96	+20.00 = +5.98 +14.00 = +5.94	+20.00 = +7.97 +0.02 ×H +16.00 = +7.93 +0.02 ×H	+20.00 = +9.97 +0.06 ×H +18.00 = +9.95 +0.06 ×H

* Numbers above the underlined numbers in all tables represent the Wollaston corrected-curve series; those below correspond to Ostwald. The paired numbers under each part of the table give more exact interpolations for front surface power and effective edge spheric power with astigmatism eliminated.

of the lens with maximum deviation. The result is a vertical (sagittal) line focus at "SAG," much closer than the "far-point sphere" which is drawn in solid line. Rays in the vertical plane converge still more, forming a horizontal (tangential) line focus at "TAN." The dioptric distance from the "far-point sphere" to "SAG" is the gain in spheric power, while the distance from "SAG" to "TAN" is the gain in plus astigmatism, axis horizontal.

The result is an effective power through the margin of such a lens 30 degrees from the principal axis of plus 9.36 with plus 5.32 diopters astigmatism axis 180 degrees (in figures and tables, axis horizontal is abbreviated as "H," vertical "V," axis as "X").

Figure 5 shows other examples of changes in sphere and the induction of astigmatism in vision 30 degrees from the center of spheric minus 6.0D. and plus 4.0D. lenses, due to choice of lens shape. These are true scale drawings. The total range of astigmatism change in gaze wandering around the lens margin is twice that given, because the axis changes 90 degrees in gaze moving from upward to sideways. In the biconcave lens "A," the figures in the frontal view show that marginal power and astigmatism changes would be intolerable.

The same central power in a lens of meniscus form reduces the astigmatism, but also causes loss of spheric power. Lens "D" with a front surface of plus 3.0D. best corrects the marginal astigmatism. In the plus 4.0D. spheres, the best front surface is found in "G" which has a front power of plus 10.5 diopters. Notice the loss of spheric power. In lens form "H," the astigmatism changes axis. In strong plus lenses, better correction can be obtained in the lenticular form, "I," because the center thickness can be reduced.

In Table 1 are figures calculated by Swaine[3] (1938) showing the effective power and marginal astigmatism in gaze elevated 15 degrees from the lens center. (I have transposed tangential and sagittal power into familiar prescription form.) The vertical column at the left of the table shows the range of front surface powers in diopters for each of the lens powers from minus 22 through plus 10 diopters shown along the lower horizontal border.

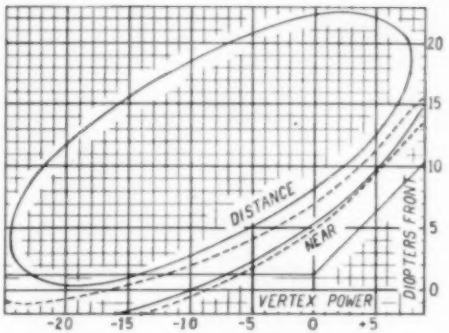

Fig. 6 (Miles). The Tscherning ellipse shows the front surface curve required to eliminate marginal astigmatism for oblique gaze in infinitely thin lenses in distance vision. The lower solid line was calculated for the reading distance.

The table is analogous in form to the Tscherning oval, Figure 6. The ocular surface curves are not given, but must be appropriate for the vertex power desired. The underlined figures indicate the approximate best curves, but the necessary interpolations for best curves in the Wollaston and the Ostwalt form are given in pairs under each column in the figure.

Tscherning[4] (1908) discovered that the Wollaston curve is continuous with that of Ostwalt[5] (1898) to form an ellipse (fig. 6). Front surface power is arranged in vertical columns for the vertex powers in the horizontal. Vertex powers below minus 24.68 diopters or above plus 7.88 diopters cannot be fully corrected, except by aspheric grinding. Gullstrand[6] (1911) designed these lenses for strong hypermetropia or aphakia, and Zeiss manufactured them under the name "Katral." In 1917 they cost $55.00 a pair.

Tscherning's ellipse was calculated for infinitely thin lenses and infinite object distance. His curve for object distance of 33 cm. has front surfaces about 2.5D. flatter and is shown in the lower solid line of Figure

6. When actual lens thicknesses are introduced into the calculations, the front surfaces are about one diopter flatter for object distance infinity and 0.50 diopters flatter for near, as shown by Percival[7] (1901, 1934), (the broken lines in Figure 6). Modern lenses are still larger in diameter than those in 1934, requiring a center thickness about 1.5 mm. greater. This would lower Percival's curve an additional 0.25 to 0.50 diopters on the right end.

Swaine[3] (1938) considered that people restricted careful gaze to within 15 degrees of the center of their spectacle lenses. His figures in Table 1 are therefore for only 15 degrees of oblique gaze. This restriction may be true in those long habituated to glasses, particularly if those glasses blur the vision through lens margins.

Modern spectacle lenses are becoming still larger in diameter, so that glasses will restrict eye movements as little as possible. The automobile driver needs to hold his head straight ahead, while the eyes move to check traffic and the instrument panel. The desk worker moves his eyes from typewriter to notebook to letter basket and back to typewriter.

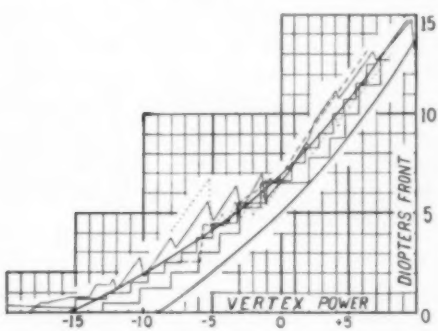

Fig. 7 (Miles). The smooth curves of Percival for distance and near are compared to the surfaces for five single vision corrected-curve series on the present market. The steplike graphs indicate the base curves of two manufacturers who maintain the same front surface for several vertex powers. The sawtooth graphs indicate constant rear surfaces for several vertex powers.*

* Since this paper was written, the lens series indicated in Figure 7 by the sawtooth dots, which are far from the corrected line, has been improved.

I believe Swaine's figures in Table 1 should be altered to cover oblique gaze to 30 degrees, which would make them even more convincing as to the value of corrected curves. Each deviation in sphere and in cylinder from vertex power should be multiplied by eight. That this factor is about right is readily seen by reference to Figure 4, the broken lines.

The solid horizontal line in Figure 6 which turns up 45 degrees in plus lenses shows the position of the cheap meniscus lens on a base of 1.25 diopters. Reference to Table 1 shows that marginal astigmatism can amount to 0.06 diopters in as weak a lens as one diopter. Meniscus lenses on a base of three or six diopters are superior for plus powers, but in certain powers the astigmatism may exceed 0.12 diopters.

Most of the single-vision corrected-curve lenses on the American market are produced by five companies. Although approximately 85 percent of the lenses being worn today were finished on both sides at the factory, some must have the second surface finished in a local grinding laboratory. There may be in some cases occasional substitution of base curves when the proper blank is not in stock, but this error is probably not important, or common.

In Figure 7 are compared the smooth curves of Percival for distance and near correction, with the five corrected curve series on the present market. The step series indicate that two manufacturers use constant front curves for a group of different vertex powers. (When the vertex power falls on the vertical portion of a step, the front surface is indicated by the upper limit.) The saw tooth series indicate that three manufacturers use a constant ocular surface as a base curve for several vertex powers.

It can be readily seen that four manufacturers produce lenses corrected for an object distance of infinity or beyond. One series is correct for object distance about 67 cm. The series appearing above the infinity line

TABLE 2

EFFECTIVE MARGINAL POWER*

Front Surface Power	Object at Infinity	100 cm.	50 cm.	33 cm.
+1.50	−5.99−0.44×H	−5.95−0.30×H	−5.92−0.15×H	−5.90
+2.00	−5.94−0.34×H	−5.91−0.20×H	−5.88−0.06×H	−5.85+0.08×H
+2.37	−5.90−0.27×H	−5.88−0.14×H	−5.85	−5.82+0.14×H
+2.50	−5.89−0.24×H	−5.87−0.10×H	−5.84+0.03×H	−5.81+0.16×H
+3.00	−5.85−0.15×H	−5.83−0.02×H	−5.81+0.11×H	−5.79+0.24×H
+3.12	−5.84−0.13×H	−5.82	−5.80+0.12×H	−5.78+0.25×H
+3.50	−5.81−0.07×H	−5.79+0.05×H	−5.77+0.17×H	−5.75+0.29×H
+3.87	−5.79	−5.77+0.10×H	−5.75+0.21×H	−5.73+0.32×H
+4.00	−5.78+0.01×H	−5.76+0.12×H	−5.74+0.23×H	−5.72+0.34×H

* The effective marginal power is given for various forms of −6.0D. lenses of thickness 0.6 mm., 25 mm. from the rotation center, calculated for object distances of infinity, 100 cm., 50 cm., and 33 cm.[9]

of Percival would show a little more spheric power loss, but possibly a greater correction of marginal astigmatism. The lower step curve was designed to favor the correction of marginal astigmatism for an intermediate distance, to obtain a reasonable average amount at infinity and the reading distance.

This idea of a lens series for an intermediate object distance was first suggested by W. B. Rayton[8] (1930). To illustrate the possible importance of this modification, I have transposed and extrapolated some figures given me by Klingaman[9] which are shown in Tables 2 and 3.

Table 2 shows the marginal power and astigmatism changes in lenses of minus 6.00 diopters for front surfaces from plus 1.50 to plus 4.00 diopters. With the plus 1.50 front surface, the lens is well corrected for 33 cm. working distance, but the marginal astigmatism for objects at infinity would be 0.44 diopters. The lens corrected for object distance 100 cm. does well for infinity, but is defective for reading. A front surface of plus 2.5D corrects perfectly for an object distance of 67 cm., and appears to give the least marginal astigmatism at infinity and the reading distance. In Table 3, the same effect is shown in case of lenses of plus 4.00 diopters vertex power.

Some manufacturers point with pride to the large number of steps in their corrected-curve series. In Figure 7, it can be seen that more steps will keep a series closer to the Percival line. However, less marginal astigmatism occurs in weak powers, so the smallest steps should be on each end of the series, particularly in plus lenses. Possibly fewer steps would reduce costs without increasing marginal astigmatism.

TABLE 3

EFFECTIVE MARGINAL POWER*

Front Surface Power	Object at Infinity	100 cm.	50 cm.	33 cm.
+7.50	+3.93+0.32×H	+3.88+0.23×H	+3.84+0.14×H	+3.81+0.05×H
+8.00	+3.90+0.25×H	+3.85+0.17×H	+3.81+0.06×H	+3.78
+8.50	+3.87+0.19×H	+3.82+0.11×H	+3.78+0.03×H	+3.75−0.04×H
+8.87	+3.85+0.15×H	+3.81+0.08×H	+3.76	+3.72−0.09×H
+9.50	+3.83+0.08×H	+3.79+0.01×H	+3.73−0.06×H	+3.68−0.13×H
+9.62	+3.81+0.05×H	+3.77	+3.72−0.08×H	+3.66−0.17×H
+10.50	+3.79	+3.73−0.06×H	+3.68−0.13×H	+3.63−0.21×H

* The effective marginal power is given for various forms of +4.0D lenses, 3.72-mm. thick.[9]

Bifocal segments present no great problem, because the segment for near vision is always stronger plus. The flatter front surface required for the reading distance is neutralized by the sharper front required for stronger plus power. Segments are usually small so that marginal rays do not exist from the insert lens. Certainly marginal astigmatism would be a factor from the distance lens, because the segment is not placed in the optical center.

Evidences that corrected curves are unimportant

To this point, corrected curves have been apparently of great value, to be desired at whatever expense. In practice, these good effects are easily lost. Corrected curve lenses should be fitted symmetrically so that the average position of gaze is through the optical center and so that the visual line is perpendicular to the ocular surface. To illustrate the effect of pantoscopic tilt, Figure 8 shows the power and astigmatic changes in vision through the center and upper margin of strong biconvex and best corrected lenses.

Spectacle lenses are designed on corrected curves for a distance from the cornea of 12 mm. Figure 8, parts "E" and "F," show the effect of variations from this position, in case of a plus 10-diopter corrected lens.

E. D. Tillyer[10] (1945) suggested that, since hypermetropic eyes are shorter and myopic eyes are longer than normal, corrected-curve lenses which today are calculated for 27 mm. from the lens to the rotation center of the eye should be designed differently for highly ametropic eyes. He suggested that in strong plus lenses, the 27-mm. distance be changed to 24 mm. and in strong minus lenses to 30 mm.

A difference of two mm. in this distance would change marginal astigmatism 0.04 diopters. Unfortunately, in normal eyes, the distance from the cornea to the rotation center varies from 10.5 to 15 mm., and the range is equally great in high myopia or hypermetropia. Adding variations of position of the eye in the orbit, the 27-mm. lens to rotation center of present corrected curve lenses must do for variations anywhere between 15 and 38 mm.

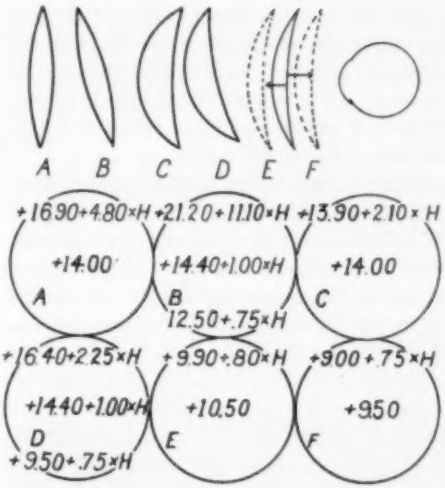

Fig. 8 (Miles). There is enormous marginal error in a biconvex plus 14-diopter lens "A," which is exaggerated by the usual 15-degree pantoscopic tilt, "B." This occurs even in the best corrected curves, "C" and "D." The changes which occur in moving a strong lens from its normal 12-mm. distance are shown in "E" and "F."

There is strong evidence that most people do not object to marginal astigmatism. Most persons do not notice changes in base curves, say from cheap meniscus to corrected curves, or changes to a different brand. I have had one patient who objected to minus 4.0D. corrected-curve lenses who had been wearing the same power in Zeiss Punktals. The front surface had a difference of 0.75 diopters.

Some patients may prefer lenses which produce marginal astigmatism. Since 1931, about 2,000 patients a year have been fitted with aniseikonic glasses in which one lens is made thicker and of steeper front curve. Published studies of case reports from about six different clinics agree that about 70 percent of these patients were quite pleased with these "poor quality" glasses of uncorrected curve.

Refractionists or opticians may freely dis-

regard the specifications for corrected curve by calling for a deeper curve to clear the patient's eyelashes. Some request that minus lenses be made paper thin to reduce weight. Some will match the front surfaces of the two lenses for cosmetic reasons.

That manufacturers do not practice what they preach is most evident in their neglect to design corrected curves for those patients who have astigmatism. The power required in one meridian may call for a front surface curve of plus 6.0D., while the power in the other calls for plus 10D.

To design corrected curves for both meridians would require bitoric lenses. Such lenses would have toric or astigmatic surfaces on both sides, and are considered at present not worth the trouble and expense (except in aniseikonic glasses). At present, an average base curve for the two meridians is taken, giving a poor correction in both. A patient with astigmatism must either accept the marginal error produced by his glasses, or learn to use only the central portions of his lenses.

It seems illogical that the manufacturers should so highly recommend corrected curves for spheric refractions and make no attempt to design similar correction for sufferers from astigmatism.

Summary and Conclusions

Quality in a spectacle lens may be defined as the sum of all optical characteristics which produce retinal images and ocular movements like those of a normal unaided eye. This discussion is for the purpose of explaining to the ophthalmologists such attempts which have been made to attain this goal.

Spectacle lenses with corrected curves are of real value, particularly in the higher powers. They should be made available for patients suffering from astigmatism.

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DIAGNOSIS OF METASTATIC TUMORS OF THE ANTERIOR OCULAR SEGMENT*

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Metastatic tumors of the anterior ocular segment may arise through blood-stream metastases from a primary tumor elsewhere in the body or by implantation from a neoplasm of the posterior segment. Involvement of the anterior part of the eye by blood-borne metastasis is rare, there being only 35 recorded cases in the literature. According to Greear[1] there are no more than six cases of proved metastatic carcinoma to the iris without other ocular involvement.

Although in most cases the patient with a metastatic lesion of the eye is suffering from a known malignancy, occasionally the ocular metastasis is the first sign of neoplastic disease. In such instances the differential diagnosis between a primary and a metastatic tumor of the iris is difficult. In many cases metastatic tumors may simulate inflammatory nodules or hypopyon. Three cases will be presented to illustrate these difficulties in diagnosis.

CASE REPORTS

CASE 1

This case illustrates the simultaneous involvement of iris and choroid by metastatic carcinoma.

E. H., a 50-year-old woman, noticed a pinkish-white mass on the iris of her left eye one morning while looking in the mirror. Her general health at this time was good except for some vague abdominal distress.

Vision in the left eye was 20/20; tension and fundus were normal. No other ocular abnormalities were found other than the small pinkish-white tumor which appeared very prominent on a blue iris. The eye was enucleated within three weeks after the tumor was first discovered. The patient died two months subsequently. Unfortunately, no information is available concerning her final illness and no autopsy was done.

Microscopic examination of the eye revealed the iris and ciliary body on one side to be replaced by a tumor mass which infiltrated

Fig. 1 (Middleton). Metastatic adenocarcinoma of iris (Case 1).

the trabeculum and Schlemm's canal in this region. The tumor was composed of large pale-staining cells containing large round or oval nuclei which varied considerably in size and staining density and contained prominent nucleoli. The cells were arranged around central lumina in crude glandular formation (fig. 1). Two small nests of tumor cells were present in the choroid, one on each side of the optic nerve (fig. 2).

The metastatic tumors in this case are adenocarcinomas, but without additional information it is impossible to state the origin of the primary tumor with exactness.

* From the Department of Ophthalmology, University of Illinois School of Medicine, and the Illinois Eye and Ear Infirmary, Chicago, Illinois. Read before the Chicago Ophthalmological Society, April, 1951.

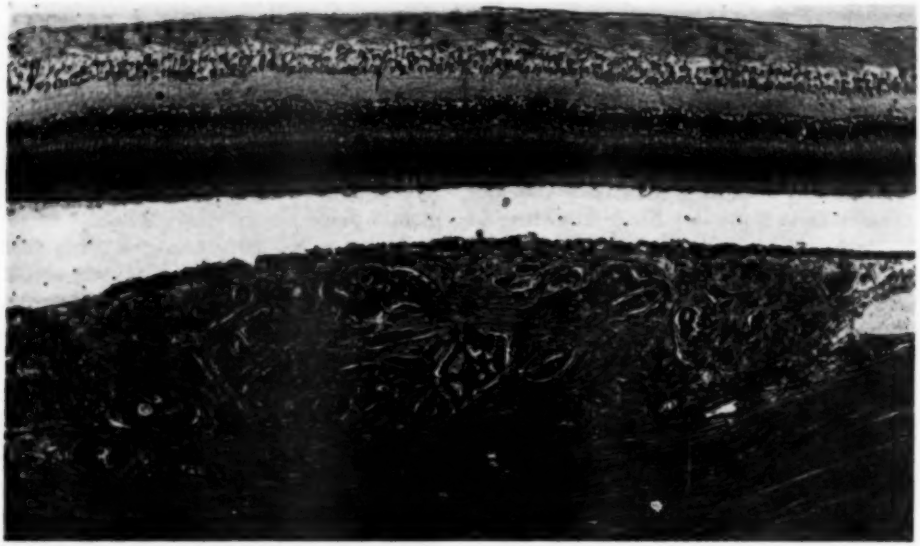

Fig. 2 (Middleton). One of the metastic nodules to the choroid (Case 1).

CASE 2

F. H., a 37-year-old man, was seen for the first time April 14, 1948, complaining of pain and tenderness in the left eye. There was no history of a blow or injury of any kind.

Examination of the eye revealed a small white nodule on the anterior surface of the iris at the 4-o'clock position near the base. The globe showed pericorneal injection and tenderness to pressure over the region of the ciliary body. Vision was 20/20. The right eye was normal.

Laboratory work-up showed a negative blood Wassermann and spinal tap, negative tuberculin skin test, and normal X-ray films of the left orbit and hands and feet. Chest X-ray studies were reported as "suspicious," although a definite X-ray diagnosis was not made at this time.

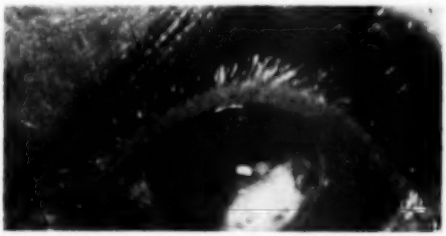

Fig. 3 (Middleton). External photograph, Case 2, showing large iris tumor. Note intense congestion of globe.

In two weeks the iris nodule had enlarged to six mm. in diameter and was touching the posterior cornea. Vision had dropped to 20/200. The sclera showed intense congestion, the cornea was edematous, aqueous ray was positive, tension was 18 mm. Hg (Gradle) (fig. 3). Transillumination showed the nodule to be solid. The fundus could be seen with fair clearness and appeared normal.

In view of the sudden onset, rapid progression, and intense inflammatory symptoms, it was felt that the iris nodule was inflammatory in nature. The patient received atropine, systemic penicillin, foreign-protein therapy, and streptomycin iontophoresis without benefit. The eye rapidly deteriorated and pain became so intense that enucleation was preformed on May 8th.

Shortly thereafter the patient developed weakness of the right arm and leg, severe headaches, and papilledema of the right disc.

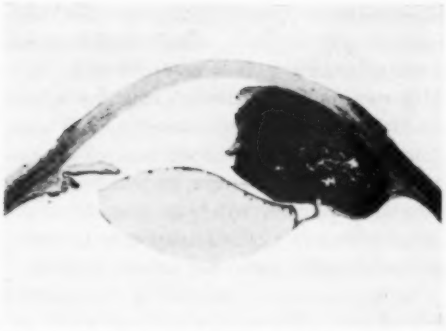

Fig. 4 (Middleton). Metastatic carcinoma of iris and ciliary body (Case 2).

X-ray studies of the chest now revealed a definite tumor of the left upper lung. The patient died on May 30th.

Microscopic examination of the eye showed that the iris on one side had been replaced by a large tumor mass composed of undifferentiated cells growing in sheets and columns (fig. 4). The tumor extended into the ciliary processes and the trabeculum. Within the tumor were large areas of necrosis containing masses of leukocytes (fig. 5).

An autopsy report, kindly furnished by Dr. Horace K. Giffen, directory of laboratories, Youngstown Hospital Association, Youngstown, Ohio, revealed a bronchogenic carinoma of the left upper lobe with metastases to the peribronchial lymph nodes and the brain. The lung tumor was characterized by moderately large cells with poorly defined cell borders, pale, finely granular cytoplasm, and vesicular, moderately large round or oval nuclei, thus corresponding closely to the cells found in the iris tumor (fig. 6).

CASE 3

This case illustrates involvement of the anterior segment by intraocular metastases from a tumor in the posterior segment. An unusual feature is the inflammatory symptoms which obscured the diagnosis.

J. B., a nine-year-old Negro, was first seen at the Illinois Eye and Ear Infirmary on May 10, 1948. Three months previously he had been kicked in the left temple, following which the eye had become inflamed and vision declined. Five years previously he had had a squint operation at Cook County Hospital.

Examination showed the right eye to be normal; left eye: vision 20/70, tension 51 mm. Hg (Schiøtz). The bulbar conjunctiva was slightly injected. The lower portion of the anterior chamber was filled with a white exudate, and there were clumps of the same material on the anterior surface of the iris, the anterior lens capsule, and floating free in the chamber. A space of about one mm. between the iris and anterior lens capsule was noted. The lens was clear. The vitreous contained floating opacities. The fundus could not be seen except for a dull red reflex.

Laboratory work including tuberculin skin and X-ray studies of the skull, orbit, and chest were all normal. Three aspirations of the anterior chamber were performed and in

Fig. 5 (Middleton). Higher-power view of tumor in Figure 4, showing massive necrosis and leukocytic infiltration.

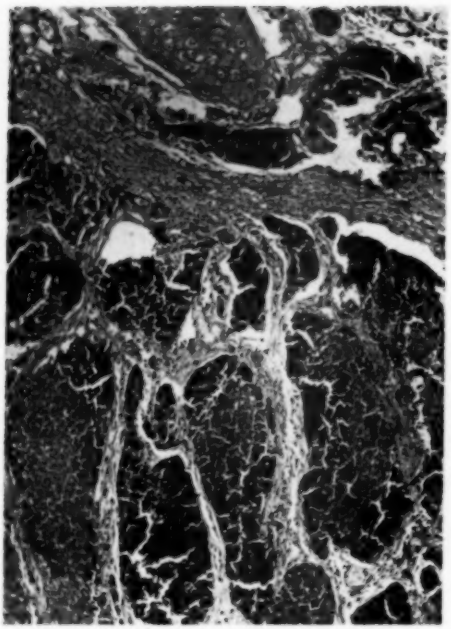

Fig. 6 (Middleton). Primary bronchogenic carcinoma (Case 2).

one instance the chamber was irrigated. Cultures and guinea pig inoculations of the aspirated material were negative. The vision of the left eye declined to light perception. Tumor was suspected, and on June 9, 1948, the eye was enucleated.

Shortly thereafter the patient developed swelling of the cervical lymph glands and biopsy of one of these showed metastatic tumor. He received X-ray therapy to the left socket and cervical regions without benefit. Before death, a metastatic lesion in the right upper humerus developed.

Microscopic sections of the eye revealed a retinoblastoma just posterior to the ora serrata, with multiple intraocular metastases. Over the iris a thick layer of tumor cells was arranged around blood or lymph spaces. From here the tumor had invaded the iris, trabeculum, canal of Schlemm, and ciliary body (fig. 7).

Discussion

Primary tumors of the iris such as malignant melanomas, epithelial tumors, and leiomiomas may be very difficult to distinguish from metastatic lesions or from each other. Metastatic nodules to the iris may be single or multiple, and present in one or both eyes. They may be located at the base of the iris or at the pupillary margin, and their size may vary from pinpoint to a large mass filling the anterior chamber. Their color may be white, yellowish-white, pink, red, brown, or gray.

In approximately one half of the reported

Fig. 7 (Middleton). Involvement of anterior segment by retinoblastoma (Case 3). Note tumor-cell hypopyon.

cases the iris nodules are extensively vascularized, a feature which McKee[2] stated was also characteristic of primary malignant melanoma of the iris. In general metastatic iris tumors grow more rapidly than primary ones and pain and secondary glaucoma are almost always present.

The dark color of most primary iris tumors is a valuable point of differentiation although a melanoma without pigment (leukosarcoma) may simulate exactly a metastatic lesion.

Cysts of the iris can usually be diagnosed by transillumination.

It is usually stated that inflammatory symptoms are rare with metastatic tumors, but in 40 percent of the 35 reported cases of blood-borne metastases to the anterior ocular segment definite signs of iridocyclitis were present, such as in the second case reported here. In several of these cases the marked iridocyclitis temporarily obscured the diagnosis.

Thus, in the case reported by Stallard[3] the patient gave a history of syphilis, the Wassermann reaction was positive, and there was a typical Argyll Robertson pupil in the right eye. The left eye showed circumcorneal injection, and a pink-buff colored mass was present in the iris root between the 9- and 11-o'clock position and contained dilated vessels on the surface. The aqueous ray was positive. Punctate keratic precipitates and broad posterior synechias were present.

The first diagnosis was syphilitic iritis and gumma of the iris. However, four weeks later the patient died of carcinoma of the lung and the iris tumor proved to be a metastatic carcinoma. Stallard states that inflammatory features are more evident in metastatic carcinoma of the iris than when the choroid is involved.

In Kreibig's[4] case the first eye symptoms were those of iridocyclitis. A strongly positive tuberculin reaction seemed to confirm the diagnosis of tuberculous nodular iritis, but the nodule later proved to be metastatic from a bronchogenic carcinoma. Kreibig called attention to the recurrent hemorrhages into the anterior chamber in this case and considered them an important diagnostic point as they practically never occur with nodular iritis.

Several cases of retinoblastoma with metastatic seeding of tumor cells into the anterior chamber resembling an inflammatory hypopyon have been reported. Walker[5] described such a case in a boy, aged four years, in whom a yellow-brown hypopyon was present (from tumor cells). The eye was painful, injected, and the tension was elevated. The lens was clear but only a dull-brown retinal reflex was present. A paracentesis and irrigation of the anterior chamber were performed.

Another case reported by von der Hoeve[6] also contained a tumor-cell hypopyon. In the discussion of von der Hoeve's paper, Dr. Friedenwald mentioned another case of retinoblastoma which was confused with tuberculous iritis, and in which the preauricular glands were enlarged due to tumor metastasis, which he stated, is very rare. This is strikingly similar to the third case presented here.

It is very unsual for carcinoma to spread within the eye by seeding, although in one of Greear's[1] cases a small nest of tumor cells was growing on the anterior iris surface, presumably engrafted there from a large metastatic carcinoma of the ciliary body on the opposite side. Goldsmith[7] has reported carcinomatous cells growing free in the anterior chamber in the manner of a tissue culture.

Of the 35 cases of blood-borne metastatic tumor involving the anterior segment reported to date, 31 were carcinoma, three were metastatic melanoma, and one a sarcoma. In approximately one third of these cases choroidal lesions were also present, and in others the iris and ciliary body were simultaneously affected. The primary tumor was known in 32 cases. Breast carcinoma accounted for 14, lung carcinoma five, cancer of the gastrointestinal tract four, hypernephroma two, malignant melanoma of the opposite eye two, skin melanoma one, sarcoma of the ovary one, and carcinoma of the thyroid, prostate, and epididymis one each. The duration of life after discovery of the ocular lesion varied from two to seven months in the reported cases.

Treatment

Of course, all treatment is palliative. In many cases enucleation is necessary for the relief of pain which is often intense. Lemoine and McLeod[8] reported the arrest of activity of a metastatic carcinoma to the choroid by

X-ray therapy. A solitary iris nodule may be removed by iridectomy if the diagnosis is in doubt. However, as in our Case 3 and other cases in the literature in which a paracentesis was performed for diagnosis of the cause of a hypopyon, this procedure apparently resulted in an extrabulbar extension of the tumor and metastases to the regional lymph nodes and the orbit. Therefore, it is doubtful whether it is ever advisable to perform a paracentesis in such cases of hypopyon in children, unless perhaps the cellular material is immediately frozen, stained, and studied under the microscope at the time of operation so that enucleation can be proceeded with immediately in case tumor cells are found.

Summary

In summary, three cases of metastatic tumors to the anterior ocular segment are presented, two arising by blood-borne metastasis from systemic carcinoma, one by implantation of cells from a retinoblastoma. In patients with tumors of the anterior ocular segment a search for a primary focus elsewhere, particularly in the breast and lungs, should be made. Such metastatic tumors either from a systemic origin or from the posterior segment of the eye may simulate inflammatory lesions of the anterior ocular segment.

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I wish to express my thanks to Dr. Georgiana Theobald who kindly made available the microscopic material on the three cases herein presented.

I wish to express my thanks to Dr. Hedwig Kuhn of Hammond, Indiana, for permission to report the second case. The completeness of her records, which include an autopsy report, and the interesting and unusual aspects of her case gave the stimulus for this paper.

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THE RAUBITSCHEK ARROW TEST FOR ASTIGMATISM

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When I first described my astigmatic test, which is based on the use of a specially constructed arrow,[1-3] and demonstrated it at the Amsterdam Ophthalmological Congress in 1929, it won interest and approval and was considered a serviceable device of high sensitiveness. Some later favorable references to the test were made by Pascal[4] and Gát.[5]

In the interval, other very delicate methods gained predominance, most of them based on the Jackson cross cylinder.[6, 7] Jackson's test has been further refined by Crisp and Stine[8] using the cross-cylinder against Verhoeff's rotating cross as a target. Lebensohn[9] proposed a modification using his "astigmometer" in which he adds to the cross an inverted V, the arms 60 degrees apart, to act as a pointer. Stine[10] agrees to Lebensohn's modifications with some reluctance, claiming that the addition of more lines complicates the simplicity of the two-line cross.

Without disparaging the other tests, I believe that the "arrow test" has these special characteristics which make it a valuable test for astigmatism.

1. Two lines only are presented to the eye to be tested. These are not straight lines, they are curves turning 90 degrees each in opposite directions, comprising thus all existing radiating lines in every position from zero to 180 degrees. The transitions are very fine from degree to degree. The chart, therefore, combines the utter simplicity of Verhoeff's two-line chart with an advantage of its own. The chart in every position indicates the direction of the axis.

2. The two black curved lines, parabolas forming the arrow, are bordered each by two white hems on a gray background (Friedenwald[11]), so that parts being out of the focus merge into the background to become more or less completely invisible according to their deviation from the axis and to the amount of the astigmatism. This enhances the contrast to the well-seen part which is *in* focus. A like contrast cannot be obtained by black lines on a white background or the reverse.

3. The two curves start parallel and end in opposite directions, perpendicular to their start. This makes the comparison of the two parallel portions (indicating the axis), since they are very near to one another, extremely easy. The black line, the so-called "shadow," passes over from one arc to the other the moment the arrow is turned off axis. Pascal, in discussing the paper of Crisp and Stine,[8] called attention to a weak point in their test, namely that the impressions to be compared were not simultaneous but had to be compared from memory. In my "arrow test" all comparisons are simultaneous.

4. Since the segments of the arcs are parabolic, they are not equivalent. At the start they are extremely flat, almost straight, then they gently curve out and at their ends are sharply curved. Therefore, on rotating the disc with equal velocity, the "shadow" changes its speed very markedly, slowly creeping over the strongly curved parts and flashing rapidly over the straight parts; or, seen in the reverse, to notice a movement of the "shadow" one must turn the disc in some position very quickly, but very, very slowly and cautiously in another position. The latter is, however, just the position where the most delicate rotation is needed for the accurate determination of the axis.

The "shadow" travels on each arc almost as far for the first 10 degrees as over the remaining 80 degrees. Thus, the sensitiveness in the critical part, near to the axis, is enhanced about seven times at the cost of the part at the other end. For the first single degree this proportion is still higher. No other chart provides such a sharp differentiation.

When the "shadow" has reached the arrow point, thus accurately indicating the axis, an exact symmetry of the halves of the "shadow" has to prevail in both arcs. Inexactness, even for a fraction of a degree, will be noted by patients who observe closely as a difference in the length of the "shadow" halves.*

5. In addition to these rather geometric peculiarities, there is another rather psychologic one. From his lifelong experience, an astigmatic patient expects that the spokes of a rotating wheel will become clearer as they approach one position (the axis or its perpendicular) and become dimmer as they move away from this position. This is so natural to him that he will not notice it unless special attention is called to it. Usually it remains in his subconsciousness. The paradoxical behavior of the "shadow" is, however, so surprising that it cannot escape his attention.

The "shadow" when first seen behind runs

* On performing the Crisp-Stine test one may compare the two lines of the Verhoeff chart with the two scales of a sensitive balance which go up and down with each slight alteration of equilibrium. This also applies to the two lines of Lebensohn's inverted V and, of course, to the two arcs of the arrow in my chart. But the most sensitive part of a fine balance is its *tongue* whose vacillations show a *tiny* overweight when the up-and-down movement of the scales is too slight to be noticed. On the arrow the long straight top is like the tongue of the balance, its vacillation being represented by the alternate lengthening and shortening of the half-shadows in the adjoining arcs. There is nothing like the tongue of the balance in any other chart.

to the top with increasing velocity, and finally jumps into it. This phenomenon, a consequence of the convergence of the curves against the periphery, contrary to all experience and expectation, works as a strong stimulus to the attention. It constitutes a particularly valuable feature of the arrow test, not shared by any other test, so far as I know.

Shortly after the last publication describing my original apparatus and method,[3] I developed a simplified procedure which made the finding of the amount of astigmatism quicker and easier. The mechanical details for making the modifications were worked out by the original manufacturing firm of Doerffel and Färber, Berlin. Owing to the generally unfavorable conditions at that time (1933), they soon discontinued manufacturing the instrument both in the old and in the projected new form. Thus the modifications of the instrument and method have remained unknown.

New appearance of chart

Instead of the former single revolving disc there are now two. The front disc or "target" disc is identical with the original single disc, bearing the same parabolic arcs with black middle stripes and white borders, starting flat and parallel and ending in divergence, thus forming a sharply pointed "arrowhead."

The modified chart has, however, a slit along the radius opposite to the arrow point, so that a narrow strip of the covered back disc appears in it. On both sides of the slit white figures are inscribed. These figures are related to drawings on the back disc which will be explained later.

The back disc or "reading" disc has been added to the former single front disc. It is the same size and is completely covered by the front disc except for a narrow strip which appears in the slit of the front disc. The "reading" disc bears numerous black curves of different thicknesses drawn over systems of blue parallel circular arcs, the significance and use of which will also be explained later.

The scale, with a double inscription of the degrees, remains unchanged. Both systems are clockwise. The inner one is for use with minus cylinders, and has the zero on top and bottom, the outer one is for plus cylinders and starts with zero on left and right.

Use of new chart

To find the axis of the astigmatism nothing has to be changed. The handles of both discs, gripped in one hand, must be rotated *away* from the side of that arc in which the eye, slightly fogged by the spheric glass, perceives a part of the black streak—the "shadow." The rotation is continued (or reversed) until the "shadow" is located symmetrically on both limbs of the arrowpoint—"equality of shadows." The arrow now points to that degree of the scale which indicates the position of the axis: the outer figures are for the axis of the correcting plus cylinder, the inner ones for that of the minus cylinder.

The determination of the amount of the astigmatism is also unchanged—theoretically. But in practice it is essentially simplified, so that the examiner is relieved from the use of a table and from all calculations connected with it. This mental work is taken over by the new back disc, which is in effect a calculating machine.

As soon as the axis is found, a plus or minus cylinder, according to the kind of the astigmatism (hypermetropic or myopic), of any power but preferably of a power approximating that of the expected astigmatism, is inserted in front of the eye. The spheric lens is not to be changed if a minus cylinder is used; it is to be changed in the case of a plus cylinder, after reduction of its power from the spheric glass. The cylinder is to be inserted with its axis not in the position of the newly found axis of the astigmatism, but at a certain angle to it: 10 degrees for cases of presumably very high astigmatism, 20 degrees for average cases, 30 degrees for very low astigmatisms.

Now of course the "equality of the shadows" is gone and a new rotation of the

Fig. 1 (Raubitschek). Appearance of arrow with "shadow" (dark streak) in one limb, when arrow is not in correct position.

front disc, also *away* from the side of the "shadow," must be made in order to restore this equality. During this movement the back disc remains fixed in the position which was reached by the combined rotation of the front and back disc for the determination of the axis.

It is presumed that, in the original position of the two discs, the number of the chosen angle between the axes (20 or 30 and 10) was exposed in the slit. If not, this has to be corrected by turning the back disc 180 degrees behind the stationary front disc. Then the movement of the front disc over the stationary back disc starts. When now a new "equality of the shadows" is obtained, the true amount of the astigmatism is read off in the slit.

The big white numbers 20 on one side and 30 and 10 on the other side of the slit again signify the angle between the true and the displaced axis of the inserted cylinder. The smaller white figures mean diopters of the cylinder used. Among the parallel blue lines in the slit, the one which belongs to the concerned angle and cylinder power must be followed till the radial wire in the middle of the slit and the black curve intersected there indicate, by means of the black figures, the real amount of the astigmatism.

Only the thickest curves, corresponding to full diopters, are marked with figures. Half and quarter diopters, as well as one-eighth diopters, for the angle of 30 degrees are easily noted by their different thickness.

All the black curves are constructed according to the formula:

$$a = c \frac{\sin (180 - 2\varrho)}{\sin 2 (\varrho - \theta)}$$

The derivation of this formula with (other) theoretical considerations may be seen in my former communication.[2] In this formula:

- a equals the amount of the astigmatism in diopters
- c equals the power in diopters of the inserted cylinder
- ϱ (rho) is the so-called "guide angle"
- θ (theta) is the chosen angle of 10, 20 or 30 degrees between the axis of the cylinder and the real axis of the astigmatism

The definition of ϱ (the "guide angle") is different when a plus cylinder or a minus cylinder is inserted obliquely to the axis of the astigmatism. In the case of a plus cylinder the "guide angle" is included between the axis of the cylinder and the direction of the arrow point in "equality of the

Fig. 2 (Raubitschek). Appearance of arrow with "shadow" in the other limb when the disc has been turned too much.

Fig. 3 (Raubitschek). Appearance of arrow with equal "shadows" in both limbs at tip of arrow when the latter is in the correct position.

shadows." If a minus cylinder is used, then φ is the angle between the axis of the cylinder and the perpendicular to the arrow point in "equality of the shadows."

To illustrate this: The axis of a hypermetropic astigmatism was ascertained to be 85 degrees; inserting a plus cylinder, axis 65 degrees ($\vartheta = 20$ degrees), "equality of the shadows" re-appears when the arrow points to 115 degrees: then the "guide angle" is $115 - 65 = 50$ degrees. (The axis of the astigmatism is out of account.)

Or: The axis of a myopic astigmatism is 15 degrees, a minus cylinder, axis 165 degrees is used ($\vartheta = 30$ degrees); "equality of the shadows" reappears with the arrow pointing to 40 degrees: in this case $165 - (90 + 40) = 35$ degrees.

Since this formula had been used in my older method, there is no new principle involved in the new one. The only difference is that formerly a variable quantity, c (the cylinder power) was taken and φ was a constant. Before carrying out the test, φ had to be found from a table for each pair of axes of astigmatism and inserted cylinder. Now c (the known power of the chosen cylinder) becomes the constant and φ (about whose measure the examiner need not bother) becomes the variable. Thus the use of a table is avoided and no computation is needed on the part of the examiner. The back disc works automatically as a calculating machine, it works easily, quickly, and surely.

Because this description of the method is interspersed with various explanations and digressions, it may give the impression that the test is somewhat complicated. In reality, it is a rather simple test in practice:

1. *For the axis:*

Fog the eye slightly by overcorrecting plus sphere or undercorrecting minus sphere.

Turn the disc (both discs) *away* from the side of the arc bearing the "shadow."

When "equality of the shadow" is announced by the patient, read the correct position of the axis—for a plus cylinder from the outer scale, for a minus cylinder from the inner scale.

2. *For the amount:*

Insert a cylinder with its axis at an angle (for instance 20 degrees) to the correct axis found.

In the case of a plus cylinder deduct its amount from the plus sphere used, for a minus cylinder do not change the sphere.

Correct the position of the back disc by turning it alone (front disc fixed) for 180 degrees, if the number 20 *did not appear* in the slit at the start.

Turn the front disc alone, back disc fixed, *away* from the side of the arc with the "shadow" until a new "equality of the shadows" is achieved.

Read the correct amount of the astigmatism in the slit from the black curve which passes nearest to the point of intersection between the blue line belonging to the used cylinder and the middle wire in the slit.

In practice, some short cuts are soon developed. For instance, the examiner will generally start his subjective test after he has made an objective examination and knows approximately the kind and amount of the astigmatism. He will therefore place the

dial in the presumably correct position and make small rotations from there if necessary.

For finding the amount of astigmatism by locating the new position for "equality of the shadows," he can immediately rotate the front disc and the slit in a direction opposite to the deviation of the cylinder so that the slit exposes along the bisecting wire the black figures equal to the white figures along the edge of the slit.

To illustrate: the axis of the astigmatism is 90 degrees, the cylinder axis was deviated to 70 degrees (decreasing figures); therefore turn the slit toward 135 degrees (increasing figures). All these figures have to be read from the chart scales (for plus or minus cylinders respectively) and not from the scale of the trial frame.

If the shadows are equal in this position, then the cylinder chosen was correct. If the cylinder chosen was not correct, the disc is rotated sightly in a direction away from the limb with the "shadow" until "equality of the shadows" is obtained. The correct amount of the cylinder required is then read from the slit.

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THE SULFONAMIDES IN TRACHOMA*

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Since 1940, there has been considerable controversy as to whether or not the sulfonamides exert a specific effect on the virus of trachoma. Many ophthalmologists, notably Thygeson,[1] Sorsby,[2] and Siniscal,[3] claim that sulfonamides do have a specific effect on trachoma and actually conclude that they stand as first therapeutic choice in its treatment.

That these conclusions are not necessarily so must seriously be considered. In fact, our clinical observations extending over a period of 12 years and covering some 1,000 cases have led us to diametrically opposite views. The aim of this paper, therefore, is to state our own experiences, hoping that they will be of interest to others working on this subject.

Discussion

Prompted by favorable reports in the literature,[4-6] we first started using prontosil rubrum in 1938, and, later, sulfanilamide. Pa-

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tients with trachoma stages I, II, and early III (MacCallan), and who had had no previous treatment, were admitted to the ophthalmology department of the American University Hospital and were subjected to an intensive course of sulfanilamide by mouth.

Adults received an initial dose of 2.0 gm. followed by 6.0 gm. daily in divided doses for a period of 10 to 20 days. Children received proportionally smaller doses. In many patients we persisted with this treatment for four to five weeks and, in others, sulfanilamide administration was stopped only when the patients began to show signs of toxicity, that is, cyanosis, nausea, lowering of leukocyte count, or albuminuria.

The results with sulfanilamide were encouraging, in the fact that discomfort to the patients was lessened, photophobia and blepharospasm were improved within two days, and, locally, the conjunctival inflammation showed early subsidence and discharge was arrested. But as far as the main issue was concerned our results were truly disappointing.

The same disappointment was expressed with the results of treatment of Egyptian trachoma with sulfonamide drugs by Wilson[7] and we heartily concur with his views. Likewise Smith and others[8] and Sory[9] have expressed similar opinions.

In our cases (100 in all), the initial improvement observed was limited to cases with secondary bacterial conjunctivitis. The acute conjunctivitis having subsided, no further improvement was detected in the conjunctival papillae or the corneal pannus. Even this initial favorable effect was absent in cases not showing concomitant secondary conjunctivitis to begin with.

With the introduction of sulfathiazole, and later sulfadiazine, new hopes were entertained. We chose the following routine: (1) Sulfathiazole (five-percent solution) used locally as drops every two hours in the conjunctival sac, and (2) sulfathiazole (or sulfadiazine) given internally, six to 12 tablets per day (0.5 gm. tablets) for seven to 10 days (adult dosage), both given concurrently. The dose for children depended on age and weight. From 1943 on, sulfadiazine was used exclusively because it was less toxic and presented fewer side effects than sulfathiazole.

We continued employing this method of treatment until 1948, but the results were essentially those obtained with the use of sulfanilamide alone. Most of the patients treated with the sulfonamides alone during the period of 1940 to 1945 now returned to the out-patient department with various complaints. Some came seeking relief from trichiasis, but the majority complained of what various observers call "acute trachoma," the same condition for which they had been treated by us previously.

The course of sulfonamide which they had been subjected to did not seem to have changed the course of the disease. All patients showed a more extensive pannus than when first seen, and if the conjunctival papillae were less or smaller, it was because scar tissue had replaced them.

Sulfonamides were again given the benefit of the doubt, however, and all patients returning with photophobia, blepharospasm, conjunctival discharge, papillae, pannus, and diminished vision were given a 10-day course of six to eight sulfadiazine tablets per day (0.5 gm. tablets) together with two hourly local instillations of 20-percent sodium sulfacetimide solution. In other words, a course similar to what they previously had had was repeated, except that, after 1946, we supplanted five-percent sulfathiazole by 20-percent sulfacetimide for local instillation.

The results of this second course of treatment were in no way different from the results of the first course, except perhaps in the fact that the medications were better tolerated. The course was found to be very efficacious in the symptomatic treatment of trachoma. There was a rapid subsidence of

itching, burning, photophobia, redness of conjunctiva, and discharge.

Some cases failed to improve and on culture the conjunctival sacs were found to harbor sulfonamide-resistant organisms such as N. influenza, E. coli, and P. morgani. It was evident, therefore, that the sulfonamides were acting merely on the secondary invaders. Improvement depended on the type of the organism secondarily invading the conjunctival sac rather than on the stage or activity of the trachomatous process.

More recently a quantity of a sulfonamide known as Lutazol[10] or G33 (0.5 gm. tablets) and a collyrium of one-percent ophthazol were made available to us for experimental purposes by the League of the Red Cross Societies operating in Lebanon. These drugs were used the same way as we had used sulfadiazine tablets and sulfacetimide solution and the results obtained were similar.

Conclusions

The theraptuetic effect noted from the use of the sulfonamides consists in rapid subsidence of all signs and symptoms attributable to the concomitant acute conjunctivitis, namely photophobia, blepharospasm, spastic entropion, conjunctival injection, and discharge. These beneficial results are obtained after seven to 10 days of therapy, so that treatment with the sulfonamides for a longer period seems superfluous and unnecessarily risky.

After the acute condition has subsided, it is sometimes noted that very early pannus formations have partially resolved. This apparent improvement is due, however, to the general decongestion of the conjunctiva, improvement in circulation, and easier emptying of the capillaries forming the bed of the pannus, rather than to any specific therapeutic action of the sulfonamides on the pannus. A well-formed pannus will not respond at all to treatment of any kind. If, as is claimed, sulfonamides are specific against trachoma, they should have the same effect on a pannus regardless of its density or extent.

Again, in corneal ulceration, abatement of acute symptoms can be expected. But this response is only so far as the acute conjunctivitis itself improves. Control of secondary infection is the basis of all subsequent improvement. Also, treatment with sulfonamides minimizes postulcerative opacifications in exactly the same way as they or the antibiotics would minimize scarring, secondary infection and disfiguring following trauma to any other part of the body. Therefore, our experience permits the conclusion that sulfonamide drugs will not specifically heal corneal ulcerations coincident with trachoma. It is superfluous to mention that sulfonamides have no effect whatsoever on established leukomas.

Reduction in size, paling, and flattening of the conjunctival papillae following treatment with the sulfonamide drugs can all be explained on control of secondary infection with subsequent reduction of inflammatory signs and turgescence. A solution of 1:1,000 epinephrine hydrochloride instilled several times in the conjunctival sac will temporarily produce the same effect. Conversely, if the conjunctival sacs become reinfected some time after the cessation of sulfonamide therapy, the conjunctival papillae regain their previous size and state of congestion. When there is no secondary infection to begin with, papillae never change their appearance no matter how much or how long sulfonamide therapy is administered.

Considering all these facts, we have now adopted the following routine treatment which, in our hands, yields optimal results:

1. Grattage or expression with Knapp's forceps. This is done once at the beginning of the course of therapy. It is sometimes necessary to repeat the procedure.

2. One- to two-percent silver nitrate applied to the everted lid three to six times per week for a period of three to six months. The majority of patients tolerate this form of treatment well. For those who cannot tol-

erate it, a drop of two-percent cocaine hydrochloride or one-percent pontocaine hydrochloride instilled into the conjunctival sac a few minutes before the silver-nitrate application will make the latter procedure completely painless. Argyrosis is a very rare complication when silver nitrate is administered this way. Many observers condemn silver-nitrate therapy as being too strong and harsh. It must be remembered, however, that trachoma is a potentially blinding disease, and therefore no reagent should be considered too strong or too harsh in its treatment. Copper-sulfate stick applied two to three times per week may be used in late stages and in cases developing argyrosis.

3. Sulfadiazine, with or without local sulfacetimide collyrium, is given in adequate dosage internally by mouth for seven to 10 days. This controls secondary infection, and makes the disease itself more readily treated with silver nitrate and the period of recovery is thereby shortened.

Cases of ulcerative keratitis coincident with trachoma are treated on the same general lines, supplemented by the use of hot moist compresses of boric-acid solution, atropine, cautery, and foreign-protein therapy.

Trichiasis needs surgical correction.

Summary

Our observations on trachoma cases treated with various sulfonamides have extended over a period of 12 years.

The sulfonamides, we believe, with the exception of certain antibiotics, are probably superior to all other therapeutic agents in combating secondary infection of the conjunctiva incident with trachoma.

By controlling secondary infection, the distressing symptoms are relieved and the disease itself becomes more readily treated with grattage and silver nitrate.

Never, however, have we seen a well-developed case of trachoma cured or arrested by sulfonamide therapy alone.

Mechanical treatment followed by a course of silver-nitrate applications stands as first therapeutic choice in the treatment of trachoma. Sulfonamides are important adjuvants but should never be used alone.

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THE USE OF FURACIN IN OPHTHALMOLOGY

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Considerable progress in the treatment of infections involving the external portions of the eye and adnexa has been made by the introduction of sulfonamides and antibiotics. However, the occurrence of sensitivity reactions, coupled with a certain percentage of failures and the fairly rapid loss of potency of topically applied antibiotics, makes the availability of other effective therapeutic agents most welcome. It behooves the clinician, therefore, to become familiar with commonly known drugs in the event that circumstances warrant changing medication during a course of treatment.

With this in mind, the following report deals with the local use of furacin in treating such infections during the past two years.

PROPERTIES OF FURACIN

Furacin, a proprietary brand of nitrofurazone, is well known to the general surgeon and dermatologist, as indicated by the numerous articles regarding its use published in the current literature.[1] By the same standard, the ophthalmologist is somewhat unfamiliar with its potentialities—a few articles referring to the use of nitrofurazone in ophthalmology.[2-5]

Furacin is a yellow crystalline substance, slightly soluble in water, which has been shown by Dodd and Stillman[6] to be bactericidal for a variety of gram-positive and gram-negative bacteria. Ophthalmic preparations are a solution containing furacin (0.02 percent) and an ointment of 1.0-percent concentration, both of which retain antibacterial activity without loss of potency for at least one year without refrigeration.[7]

The exact formula of furacin ophthalmic liquid is interesting:

Furacin	0.02
Phenylmercuric acetate	0.002
Methocel 4,000	0.5
Sodium chloride	0.9
Distilled water	98.578

An antibacterial spectrum which may be of interest to the ophthalmologist is listed in Table 1. Admittedly, this is not a comprehensive list, merely including organisms which are commonly recovered from external lesions of the eye and lids.

TABLE 1

THE ANTIBACTERIAL SPECTRUM OF FURACIN IN VITRO[7]

(Bactericidal concentration in mg. per 100 cc. of broth)

Gram-negative	
Aerobacter aerogenes	4.0
Escherichia coli	1.25
Klebsiella pneumoniae	2.0
Neisseriae gonorrhoeae	1.0
Proteus vulgaris	10.0
Gram-positive	
Corynebacterium diphtheriae	4.0
Diplococcus pneumoniae	6.6
Micrococcus pyogenes var. albus	1.0
Micrococcus pyogenes var. aureus	1.25
Streptococcus pyogenes	10.0

PATIENTS TREATED

Spectacular improvement in a severe pyoderma involving the lids, which was treated with furacin soluble dressing, prompted this investigation.

For a period of one year, clinical observations were recorded with no bacteriologic investigation being done. The patients for the most part were encountered in private practice. A small minority was treated in various clinics and dispensaries. The results were sufficiently favorable to warrant a correlation of bacteriologic findings with the clinical diagnosis and response to treatment.

Altogether, a total of 596 patients were treated with furacin, either in solution or

TABLE 2

PATIENTS TREATED

Acute conjunctivitis	68
Blepharitis and blepharoconjunctivitis	59
Chronic conjunctivitis	71
Corneal ulcer—limbal and central	36
Corneal abrasions and thermal burns	83
Chalazion—including postoperative treatment	46
Acute hordeola	18
Acute dacryocystitis	8
Corneal rust deposits (after removal)	162
Laceration of conjunctiva	10
Rosacea keratitis	4
Keratitis of various types	17
Dendritic ulcer of cornea	9
Herpes zoster	3
Sjogren's syndrome	1
Infected socket following enucleation	1

ointment or both. Bacteriologic studies were done in 205 cases.

Table 2 indicates the variety of cases treated, while Table 3 lists the organisms recovered by culture from the lesions prior to treatment. Space does not permit actual correlation of the organisms with the disease.

Smears were made occasionally, stained both by Giemsa and Gram techniques. After preliminary failures to recover inclusion bodies from conjunctival scrapings, this study was abandoned. In general, organisms found in smears corresponded to the results obtained by culture. No attempt was made to study viruses.

METHOD OF ADMINISTRATION

No fixed routine of administration of furacin was followed. The frequency and method of application were governed by the findings of the individual condition to be treated. The solution was instilled according to the severity, ranging from every half hour to four times daily, after irrigation of the conjunctival sac if discharge was present. Ointment, when used, was generally at bed time unless in association with a dressing.

Massage of the lids to evacuate the contents of the meibomian glands was routine in treating blepharitis. Other forms of therapy were employed when indicated, such as warm compresses, atropine, and so forth.

During the course of the investigation, the question was raised whether the preservative, phenylmercuric acetate, played a role in therapy. As a control, 39 patients with varying conditions were treated with the solution free of the phenylmercuric acetate. The results were substantially the same as for the entire series, indicating that the furacin and not the preservative was the effective ingredient.

TABLE 3

BACTERIA RECOVERED IN CULTURES

	No. of Patients
Micrococcus (Staphylococcus) pyogenes aureus	58
Micrococcus species (several)	83
Streptococcus hemolyticus	10
Diphtheroids	15
Hemophilus conjunctivitidis (Koch-Weeks bacillus)	4
Pseudomonas aeruginosa (B. pyocyaneus)	3
Aerobacter aerogenes	2
Diplococcus pneumoniae (Pneumococcus)	4
Coliform organisms	3
Bacillus subtilis	3
Moraxella lacunata	3
No growth	17

(Mixed flora in several instances.)

RESULTS

All traumatic lesions involving the cornea and conjunctiva healed rapidly and without any signs of infection. It is believed that furacin has no inhibitory action on epithelial regeneration of the cornea. As indicated in Table 4, it is apparent that furacin has a beneficial effect in bacterial infections involving the cornea, conjunctiva, and lids, but is of limited or no value in those conditions

TABLE 4

RESULTS OF TREATMENT IN NONTRAUMATIC CONDITIONS

	Recovered or Improved	*No Improvement*
Acute conjunctivitis	51	7
Blepharitis with conjunctivitis	56	3
Chronic conjunctivitis	58	13
Corneal ulcer	34	2
Acute dacryocystitis	8	0
Keratitis—various types	14	3
Virus infections	0	12
Sjögren's syndrome	1 (slight benefit)	0
Infected socket	1	0
Other lid infections (hordeola, etc.)	15	3
Total	248—85%	43—15%

which are currently accepted as being caused by a virus.

As is to be expected, the response to treatment was more gratifying in the acute infections and less so in chronic conjunctivitis not associated with blepharitis. This same observation is true for all other forms of therapy.

Most of the patients with chalazion required surgical treatment. In this instance, the medication was employed postoperatively, with smooth and rapid recovery. It is not implied that furacin obviates the necessity of surgery when indicated.

Five patients exhibited signs of hypersensitivity to the drug, all in ointment form. One patient gave a history of angioneurotic edema, reacting to numerous drugs in violent fashion. The others had mild edema, itching, and discoloration which subsided promptly upon cessation of the drug. No side effects were noted in those patients treated with the solution.

Comment

Clinical observations indicate that furacin is an effective antibacterial drug which may be safely prescribed for a variety of conditions involving the external eye and lids. It compares favorably with currently prescribed drugs in regard to stability, efficacy, and antibacterial spectrum. Most patients experience a pleasant sensation after its administration in solution, due most likely to the methyl cellulose. This latter perhaps accounts for the slight benefit in the one patient with Sjøgren's syndrome.

Its routine use is recommended in the therapy of superficial corneal injuries, since epithelial regeneration evidently is not inhibited. The low index of sensitization is also a desirable feature in such therapy.

The results obtained in this study suggest that furacin has a definite place in ophthalmology, and that general usage will soon prove its worth.

Summary

1. Furacin, in the form of ophthalmic liquid and ointment, has been employed in treating 596 patients with a variety of conditions involving the external eye and lids.

2. Bacteriologic studies were done in 205 patients, cultures of whom yielded micrococci (staphylococci) in the majority of cases.

3. Prompt healing without infection resulted in all traumatic lesions treated.

4. Recovery or improvement was observed in 248 patients who were considered as having an infectious lesion, excluding those with chalazia. This amounts to 85-percent benefit.

5. A low incidence of allergic phenomena was observed in the patients treated—all occurring in patients treated with the ointment which has a concentration considerably greater than the liquid. Duration of contact may be a factor also, as the solution naturally dissipates more rapidly.

6. Little or no benefit was observed in virus infections.

88 West Utica Street.

I wish to thank Dr. Wilbur Spencer who collaborated in most of the bacteriologic investigation. Funds for the investigation and supplies of furacin were kindly furnished by The Eaton Laboratories, Norwich, New York.

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ELECTRORETINOGRAPHY: A CLINICAL SURVEY*

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AND
JAMES MILES O'BRIEN, M.D.
Bridgeport, Connecticut

The discovery of the electrical potential produced by the action of light upon the retina by Holmgren[1] in Sweden and Dewar and McKendrick[2] at the University of Edinburgh, in 1865, has led to the development of the recording and measurement of this phenomenon, now known as the electroretinogram. Much has been learned about vertebrate and invertebrate electroretinography since the early work.

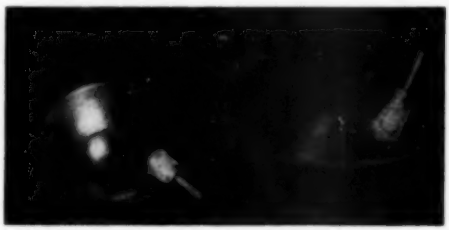

Fig. 1 (Jacobson and O'Brien). Silver-silver chloride electrode supported in contact lens.

A great part of our present knowledge of the field has been due to the work of Ragnar Granit,[3] who has studied the phenomenon extensively during the past decades at the Nobel Institute in Stockholm. Refinements of the method, such as the use of a contact lens to support the electrode, as devised by Riggs[4] at Brown University, have recently eliminated most of the difficulties of application of the method to human eyes. The development of a compact clinical apparatus for human electroretinography by Karpe[5,6] has made this study easily adaptable to clinical ophthalmology. A review of the literature on the subject has recently been published by Jacobson and Berens.[7]

* From the Departments of Research and Ophthalmology, New York Eye and Ear Infirmary. This study was aided by a grant from the Ophthalmological Foundation.

Fig. 2 (Jacobson and O'Brien). Apparatus in use.

This apparatus has been in use recently at the New York Eye and Ear Infirmary and an initial survey was begun in order to determine the clinical uses of the machine. Both eyes of 70 patients, a total of 140 electroretinograms, have been recorded and studied. This preliminary survey has not been limited to any specific type of eye or disease process, both normal and pathologic material being included.

The apparatus employs a contact lens with a sleeve, through which a wire is led to a silver-silver electrode. Two sizes of lenses are available, and all patients examined to date have been examined with these routine glasses. The electrode in the contact lens serves as the pickup or different electrode, and indifferent electrodes are placed on the patient's forehead and in his mouth. The potential produced in the eye is led through a high gain electronic amplifier into a string galvanometer, and the record is made photographically. A photoelectric cell, connected to a second string galvanometer, records the stimulating flash of light (figs. 1 and 2).

For purposes of simplicity and brevity, the electroretinograms may be said to consist of three main components (fig. 3). After a latent period of 0.04 to 0.06 seconds, a small negative a-wave is initially recorded, followed by a sharply positive b-wave. On prolonged stimulation a more gradual wave

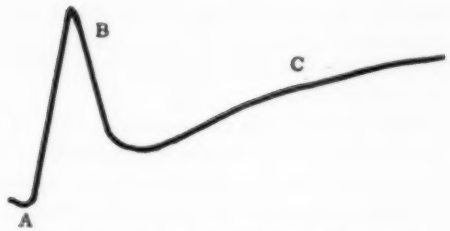

Fig. 3 (Jacobson and O'Brien). Electroretinogram schematized (after Granit). The c-wave is not produced in response to short stimuli of the order used in this study.

of positivity, the c-wave, is developed. Clinically, the amplitude of the b-wave has come to be thought of as a guide to retinal function. The normal value for the amplitude of the b-wave is approximately 0.35 mv.

In operation, the patient lies comfortably on a couch and fixates a dim orange light. After a period of dark adaptation, the voltage to the light is increased, so that the brightness is sufficient to produce a response, the period of illumination in this study being 1/25 second. It is important to reassure the patient and instruct him carefully not to move his eyes or lids during the test. Karpe uses a spring speculum to minimize lid motion, but we find this unnecessary and annoying to the patient. Pontocaine anesthesia is used routinely. Some patients were unable to attain sufficient muscular relaxation and it was impossible to obtain a conclusive tracing in these cases.

In one case, however, in which it was felt that the electroretinogram was of particular interest, an O'Brien type of akinesia was used with success. In several other cases, repeated trials were needed to obtain a satisfactory record. In general, it is felt that the method can be successfully applied in 95 percent of individuals.

The cases which comprise this series can be divided into normal and abnormal eyes. The normal eyes will be considered primarily and tracings as recorded from 10 normal eyes are shown in Figure 4. Some variation in amplitude of b-wave and duration of potential may be noted, in keeping with earlier reports. The average height in the series is 0.30 mv.

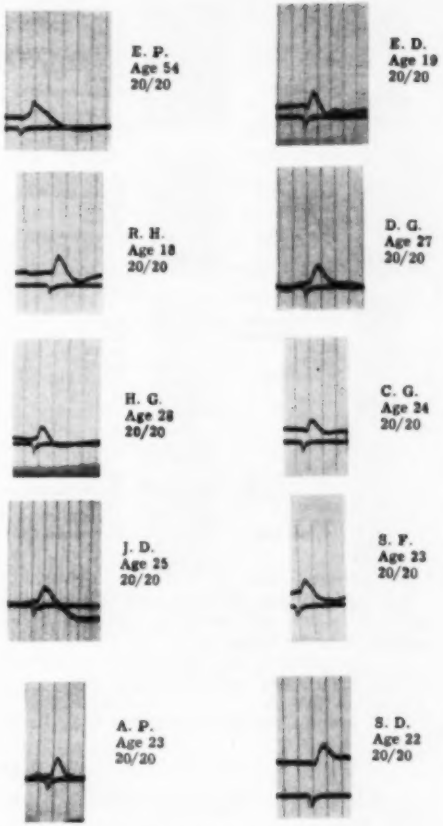

Fig. 4 (Jacobson and O'Brien). Normal electroretinograms from normal eyes. Time interval between heavy vertical lines is 0.1 second. Lower line indicates stimulus; upper line indicates response.

Eight pathologic cases are presented in Figure 5. The case of retinitis pigmentosa shows what has been termed by Karpe "extinguished electroretinogram"; the case of retinal detachment "subnormal electroretinogram"; and the patient with retrobulbar neuritis shows a "supernormal electroretinogram." These terms refer to the height of the b-wave. The other diseases do not produce abnormal electroretinograms.

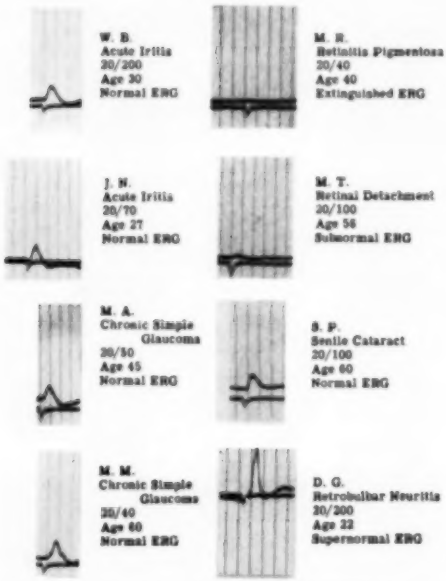

Fig. 5 (Jacobson and O'Brien). Electroretinograms from eyes with pathologic conditions. Time interval is as in Figure 4.

Summary and Conclusions

The human electroretinogram may be accurately and easily recorded in clinical surroundings by the use of the apparatus described. Few technical difficulties are encountered and it is quite possible to survey large clinical groups with the machine. The future clinical value of the method is to be determined. Further studies are in progress to show the findings in pathologic conditions of the retina. A survey has also been initiated to determine the value of the procedure in determination of retinal function prior to cataract surgery.

By virtue of the method's ability to test retinal function in an eye which does not permit observation of the retina, we feel that there are definite potential applications of the method. It has been pointed out by Karpe that the method is not a test of macular function, and this is a limiting factor in its application.

We wish to express our appreciation to Dr. Conrad Berens, without whose encouragement and coöperation this work could not have been undertaken.

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DETECTION OF INTRAOCULAR SPECTACLE-GLASS FOREIGN BODIES*

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The ophthalmologist frequently seeks radiologic consultation to determine whether a glass foreign body exists in the eye or its surrounding structures following the accidental shattering of spectacles. Interestingly enough little has appeared in the literature to date, and most textbooks of ophthalmology fail to mention whether spectacle glass is radiologically visible.

Inasmuch as the shattering of glass is not an uncommon accident and the question of the presence of intraocular foreign body frequently arises, we have been stimulated to investigate the problem. In resolving this problem two important features must be considered: (1) The nature of the glass foreign body—that is, its radiovisibility, which is determined by its size and chemical composition, and (2) the roentgen technique to be employed.

Since the opacity of any material to X-radiation is proportionate to the atomic weights of its constituents, it follows that glass containing heavier elements, such as lead or barium, will cast denser shadows than glass containing only silica and the oxides of sodium or potassium. Furthermore, the larger the foreign body, the more radiation it will absorb and the denser the shadow it will cast. The location of the foreign body is important, since substances in the anterior segment can be easily visualized by tangential bone-free films; whereas, more deeply located fragments are more difficult to detect roentgenographically.

The ingredients of ophthalmic glass are principally the oxides or salts of metals that are found in natural rock including silica, sodium and potassium oxides, nitrates or carbonates, calcium oxide or carbonate, or aluminum oxide. In addition, salts of heavy metals are used to impart special properties to the glass, for example, lead oxide (as red lead or litharge), barium oxide, or carbonate, zinc, boric, antimony, magnesium, selenium, cobalt, and nickel oxides, and fluorine as fluorides. The approximate composition of ordinary ophthalmic crown glass is given by:

ELEMENT	PERCENT
Silicon dioxide (SiO_2)	69.8
Sodium oxide (Na_2O)	8.3
Potassium oxide (K_2O)	7.5
Calcium oxide (CaO)	12.5
Sodium sulfate (Na_2SO_4)	
Sodium chloride (NaCl)	
Antimony oxide (SB_2O_3)	1.9
	100

In view of the correlation between the atomic weight of metals and their density as viewed radiologically, a listing of the metals found in lens studies is of interest:

ELEMENT	ATOMIC WEIGHT
Barium	137.36
Lead (flint)	207.22
Cerium (cruxite, soft lite)	140.13
Iron (calabar)	55.84
Silver } (noviol)	107.88
Uranium }	238.14
Silicon } (crown)	28.06
CA }	40.08
Sodium	22.99
Magnesium (used in coating lenses)	24.32
Copper (azurlite)	63.57
Manganese	54.93

PRESENT STUDY

To determine the relative radiopacity of various types of spectacle glass, lenses of identical thickness and diameter were placed on film and exposed simultaneously to a 50-K.V. roentgen beam. As predicted, the density of each shadow varied proportionately with the atomic weights of its constituents and the proportions of these constituents (fig. 1 and table 1).

Thus, flint-glass, which contains 53 percent

* From the Long Island College Hospital and College of Medicine, State University of New York.

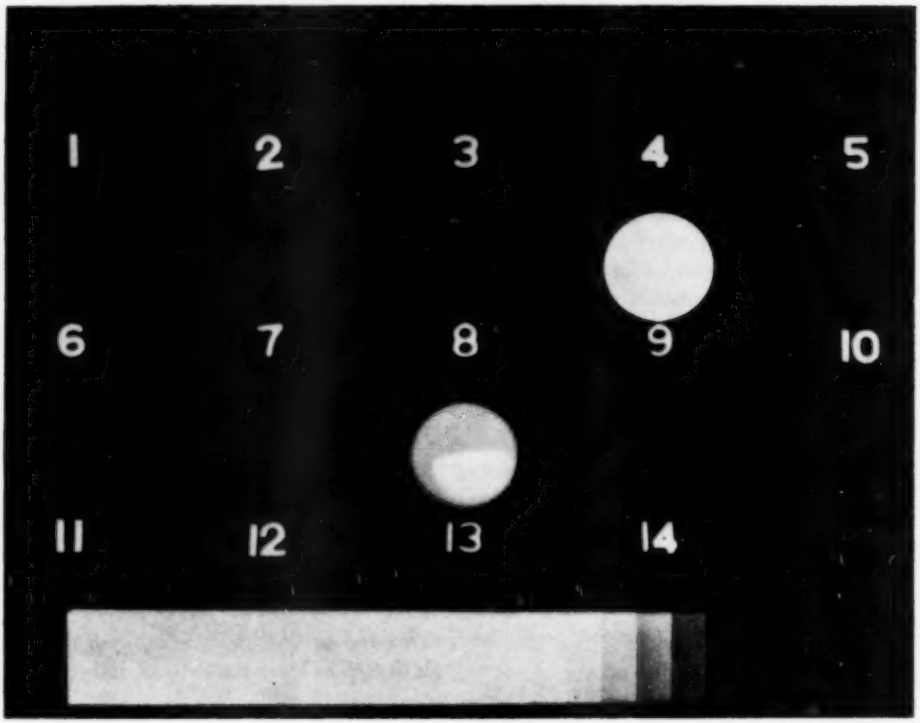

Fig. 1 (Zwanger, Gilroy, and Jones). Density to X-radiation of common lenses as compared with each other and with aluminum ladder. See Table 1.

TABLE 1

DESCRIPTION OF LENSES SHOWN IN FIGURE 1

(1)	Softlite #1	Flesh color produced by manganese dioxide
(2)	Softlite #2	
(3)	Softlite #3	
(4)	Softlite #4	
(5)	Calabar B	Green colored
(6)	Calabar C	Calabar glass has the same basic composition as regular crown, but with cerium (CeO_3) and iron in ferrous and ferric form added for ultraviolet and infrared absorption.
(7)	Calabar D	
(8)	Noviol	Yellow. Contains small quantities of silver oxide and uranium
(9)	Flint	Contains an appreciable amount of lead. The commercially used flint glass 1.70 index (high power) has 53-percent lead oxide; 1.61 index (lower power) has 42-percent lead oxide
(10)	Crown	See introduction
(11)	Azurlite #1	Blue. Prepared from copper oxide, chromic oxide, ferrous oxide.
(12)	Azurlite #2	
(13)	Orthogon with barium crown segment. Upper segment crown flint (contains lead). Barium crown, usually used in bifocal segments, in low power contains about 27-percent barium oxide and six-percent lead oxide, in higher power, 27-percent barium oxide and 20-percent lead oxide.	
(14)	Crown coated. One-half wave length of magnesium on surface of lens.	

Fig 2 (Zwanger, Gilroy, and Jones). Postero-anterior nose-chin (Waters) view of the skull. Note fragments of flint glass in region of left orbit. The particles are 0.5, 1.0, and 2.0 in diameter.

of lead (atomic weight 207.22), bifocal segments containing 27-percent barium (atomic weight 137.36), noviol containing uranium (atomic weight 238.14), and silver (atomic weight 107.88) cast dense shadows. The remaining types of glass showed only minor variations in density and were all only moderately opaque to the roentgen beam.

To determine the roentgen technique which best demonstrates intraocular glass fragments and the smallest size of fragment that could be visualized, particles of crown, crown-coated, flint, and barium glass measuring 0.5, 1.0, and 2.0 mm. in diameter were affixed to the closed eyelid or the side of the orbit with cellophane tape. Several projections were then made.

Our best results were obtained utilizing the Bucky-Potter diaphragm and par-speed screens with the nose-chin posteroanterior (Waters) projection.

Nonscreen techniques which have the advantage of eliminating screen defects and are not dependent for detail upon the screen crystal size have proven of less value than the Bucky-screen technique by reason of loss of clarity due to scattered radiation. The patient's head should be fixed with head clamps during the exposure so as to insure motion-free films.

TABLE 2

RADIOLOGIC VISIBILITY OF SEVERAL TYPES OF LENSES

Lens	Heavy Metal Content	Particle Size Visible
Crown	None	More than 2.0 mm.
Crown coated	Magnesium	1.0 mm.
Barium	Barium	0.5 mm.
Flint	Lead	0.5 mm.

Results

With good roentgen technique, foreign bodies from broken lenses made of such materials as flint and barium could be visualized even when only 0.5 mm. in diameter (fig. 2). Foreign bodies of crown-coated glass could be visualized when 1.0 mm. in diameter. Fragments of crown glass and the other types tested could be visualized only when larger than 2.0 mm. in diameter and on roentgenograms of good quality (table 2).

In any case in which an intraocular glass foreign body is suspected, it would be of great value to acquire a fragment of the shattered spectacle lens for actual roentgen test to determine its radiopacity; knowing the proper weight of the glass will facilitate the X-ray examination and help to establish a diagnosis.

Conclusions

1. Flint and barium lenses can be visualized in particles as small as 0.5 mm. in diameter with technique described.

2. The nose-chin, postero-anterior Waters projection assures the best radiologic results in detecting posterior-segment intraocular foreign bodies.

3. Anterior-segment foreign bodies offer no diagnostic problem since these can almost always be determined by tangential bone-free films.

Henry, Pacific, and Amity Streets (2).

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NOTES, CASES, INSTRUMENTS

A NEW CATARACT SCISSORS*

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A new scissors has been designed† for the enlargement of cataract sections. It is similar to the one used for round corneal transplantation and is available in both right- and left-hand curves. The shape and angulation

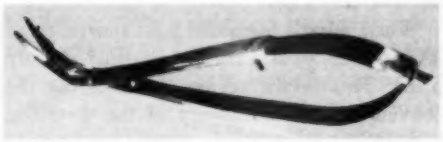

Fig. 1 (Katzin). A new cataract scissors.

of the blades has proved to be so helpful in achieving an accurately cut vertical section at the limbus that suturing is very much simplified.

A small section is made with a keratome at either end of the cataract section. The blade is introduced with the handle of the scissors vertical. This method avoids the sloping incision that results from other types of sections and allows for a more firm type of closure. It may be used either with or without a conjunctival flap.

210 East 64th Street (21).

PHOTOGRAPHY OF STRABISMUS PATIENTS USING THE POLAROID* CAMERA

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The use of conventional black and white or color photography presents some undesirable characteristics, particularly in strabismus patients. Whether special clinical reflex cameras or standard camera adaptations are employed, these cameras for average office practice require considerable time before film development and print production is complete. Hence reëxposures for desired details are inconvenient.

For some time I have employed the Land Polaroid* camera in strabismus photography with happy results. This unique camera develops a 3¼ by 4¼-inch finished print, ap-

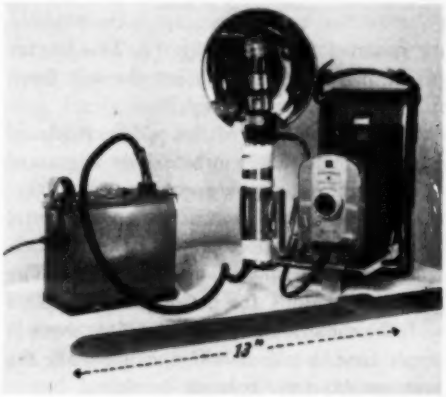

Fig. 1 (Harding) Polaroid camera, electronic flash, and holder. The easily constructed wooden "paddle" is shown.

proximately one minute after exposure. The universal photographic problems of movement, position, focus, poor lighting (including reflexes upon spectacle lenses) and mechanical failure, can thus be immediately corrected by additional exposures.

Since I found the measuring tape supplied with the accessory closeup kit for this camera very inconvenient for rapid strabismus photography, especially in the cardinal positions, I designed a simple wooden "paddle" with a crosspiece, the rear border of which is situated 13 inches from

* From the Eye Bank for Sight Restoration, Inc.

† In discussion with Dr. Harvey Thorpe of Pittsburgh and Dr. Ramon Castroviejo of New York City, it was learned that they have had similar or identical scissors made.

* Polaroid is the registered trademark of the Polaroid Corporation.

Fig. 2 (Harding). Use of paddle for focusing and centering picture.

the rounded front end (fig. 1). This border of the crosspiece fits against the left front side of the camera bed (fig. 2).

The rounded end of the paddle is placed near the right lower orbit. This alignment quickly gives a suitable focusing and picture-centering measurement. Prior to exposure, the paddle is dropped (fig. 3), and the already engaged exposure lever is pressed. The routine directions for development supplied with the camera are followed. Often there is ample time to take another picture while the previous exposure is being developed.

Fig. 3 (Harding). The paddle is lowered prior to pressing exposure lever.

For the pictures shown in Figure 4, the No. 3 accessory-copy lens was used over the camera lens, and the distance setting on the camera bed was at infinity. I have found the combined No. 3 shutter-iris setting very suitable for synchronization and exposure. The regular office lights do not interfere, although a screen to shield the patient's face from direct daylight is desirable.

The camera's development time is variable, this allows correction for any minor light intensity variations in subsequent pictures, if needed.

When using a one-sided light source, there is a tendency to have an unequal density print. This is not serious, considering the convenience and simplicity of the described equipment.

When taking a picture of a patient wearing spectacles, I found it necessary for the flash unit to be adjusted to the maximum possible height on its supporting bracket. This helps to avoid the undesirable light reflexes.

Fig. 4 (Harding). Pre- and postoperative views of the same patient. A No. 3 accessory copy lens was used over the camera lens.

The complete outfit, including the flash holder, electronic flash, and close-up lens kit, costs approximately $160.00. The total cost of a finished print is less than 25 cents each. Duplicate prints may be obtained for 12½ cents each. The diagnosis, refractive findings, basic surgical procedures, and so forth may be written upon the backs of the pictures. Small albums are available for the prints, or transparent envelopes may be used.

Eccles Building.

SURGERY FOR EXTRUDING OCULAR IMPLANTS

NORMAN S. JAFFE, CAPT. (MC) USA
Fort Campbell, Kentucky

The past decade has seen a tremendous increase in the number of integrated ocular implants inserted. The situation of active warfare has only tended to add impetus to an already well-developed trend.

Much has been written concerning the disadvantages of these implants. Various authors point to the fact that some implants of the peg type provide an avenue of infection between the brain and the exterior; they describe a continuous annoying secretion; report that a great number of implants are eventually extruded; tell how the conjunctiva thins over the tip of the completely buried integrated implant and eventually splits and how the conjunctiva over the peg type implant retracts exposing the tantalum mesh. Many feel that the ultimate results do not justify the lengthy surgery and the expense.

Many, however, hold the view that the cosmetic superiority of the so-called successful cases warrants an attempt at insertion of the integrated implant. If the complications should prove insuperable, the implant can always be removed and a plastic ball inserted. Most patients prefer to accept this risk.

It appears that the greatest motility is obtained with the peg-type of implant (Cutler, Stone-Jardon). The safest and most sterile implant is one which is completely buried. I prefer this to the peg-type. There appears to be less chance of infection and extrusion.

Not infrequently, however, the conjunctiva over the tip of the implant thins and splits in spite of the fact that Tenon's capsule and conjunctiva are closed over the implant in separate layers. The tantalum mesh is thereby exposed. Surprisingly, this does not appear to increase the chances of extrusion. The conjunctiva may continue to retract exposing more tantalum. This is most likely to occur where there has been considerable shrinkage of tissues.

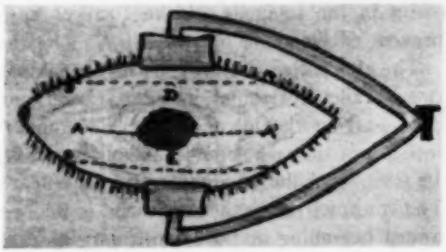

Fig. 1 (Jaffe). Diagram to illustrate technique used in surgery for extruding ocular implants.

The surgeon is left with the dilemma of removing the implant or taking his chances by allowing the conjunctiva to remain split. Undermining the conjunctiva and suturing the tear is entirely unsatisfactory since the conjunctiva at the margins of the tear is very thin for a considerable distance from the tear. Invariably the rent will recur. This has been my experience on numerous occasions.

Two techniques will be described which have been satisfactory in remedying this situation. The aim of both techniques is to utilize the abundant thick conjunctiva of the fornix (fig. 1).

From the lateral margins of the tear at the 3-o'clock and 9-o'clock positions, horizontal incisions through the conjunctiva down to the meshwork are extended for a distance of one cm. The conjunctiva is undermined. Horizontal incisions are made one cm. from the tear above and below it; that is, in the upper and lower fornix. The incisions are carried across from the medial margin to the lateral margin of the fornix. The incisions need only be deep enough to allow the conjunctiva to be drawn over the tear.

The thin margins of the tear are excised and the thicker superior and inferior conjunctival segments are sutured, using continuous or interrupted 4-0 silk. The bare areas in the superior and inferior fornix which provided the slack for the thicker conjunctiva to be brought over the tear need not be covered. The use of a conformer and an antiseptic ointment for several days will

maintain the integrity of the fornices and prevent adhesions.

The alternative method consists of making two parallel horizontal incisions about one cm. apart in the lower fornix. The upper incision of the two should be about seven to 10 mm. from the lower margin of the tear. The conjunctival segment outlined is undermined beginning at the lateral margin. The flap remains attached at the medial aspect. It is then turned up over the tear in the form of a pedicle flap. The lateral margins of the tear are sutured to the flap which is trimmed to fit the tear.

The area of the fornix from which the flap was taken should be closed with interrupted 4-0 plain catgut sutures. It may be necessary to turn down an additional pedicle flap from the superior fornix if the lower flap is not sufficient to cover the defect.

U. S. Army Hospital.

SIGNIFICANCE OF RED-LENS DIPLOPIA

Its relationship to heterophoria

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This study was undertaken in order to determine if red-lens diplopia, as uncovered in routine examinations for flying, might not be related to a heterophoria rather than a paresis of an extraocular muscle.

Tait has shown that heterophoria values for distance tend to remain constant in the average individual over a period of years.

Also, phoria values at near are normally more variable and of greater degree than at distance. It seemed, therefore, that this study would be more significant if the red-lens test was done at six meters instead of the prescribed 75 cm. At six meters, there would be less demand upon fusional vergence in order to maintain single binocular vision. Thus, the test would be of more value as a test for oculomotor palsy with the fusion factor either eliminated or kept at a minimum.

The following routine was carried out:

1. Only candidates with 20/20 vision in each eye (corrected or uncorrected) and with normal color vision were selected.

2. The candidates were then tested for heterophoria at six meters with a red, multiple Maddox rod.

3. They were next tested with a light-red lens in the primary position first and then in the cardinal positions of gaze.

4. This procedure was repeated with a blue lens.

5. The procedure was again repeated with a red lens over one eye and a blue lens over the other eye.

6. Finally, the breadth of fusion (ductions or vergences) was measured with loose prisms.

All the cases with colored-glass diplopia showed the diplopia to be of the same degree in all positions of gaze as in the primary position (two diopters variation allowed as normal). For ease in recording statistics, only figures in the primary position are given. The amount of diplopia present was measured by the minimum prism necessary for the colored images to be fused into one. The measurements were recorded and tabulated for comparison and a summary of the findings showed:

1. Of 119 cases tested 65 had esophoria, 38 had exophoria, 14 had orthophoria, and two had hyperphoria. Of the cases with esophoria, nine had over two diopters and the remaining 56 had less than two diopters. Of the cases with exophoria, seven had over two diopters and the remaining 31 had less than two diopters. The cases of hyperphoria had two diopters.

2. In the colored-glass tests, all 14 cases of orthophoria fused the three combinations of colored glass used in testing.

The remaining 105 cases of heterophoria showed:

Red lens test. Fifty-two cases fused and 53 had diplopia. Of the 53 with diplopia, 49 had the same and four had less than the amount of phoria.

Blue lens test. There were 86 cases that fused and 19 with diplopia. Of the 19 with diplopia, nine had the same and 10 had less than the amount of phoria.

Red and blue lens test. Ninety-four cases fused and 11 had diplopia. Of the 11 with diplopia, five had the same and six had less than the amount of phoria.

In the 16 cases of heterophoria over two diopters, only three could fuse the red lens, while seven could fuse the blue lens and 10 fused the red and blue lens.

Summary

Of normal candidates, 50 percent showed a red-lens diplopia. This diplopia was equal in amount to the heterophoria in 92 percent and less in eight percent; 84 percent of the candidates were able to fuse a blue lens. 90 percent were able to fuse a red and blue lens.

All the cases of colored-glass diplopia were the same in degree in the cardinal positions of gaze and, therefore, considered to be a phoria and not a palsy of an extraocular muscle.

Conclusions

1. The red-lens test acts like a red Maddox-rod test in breaking up fusion in approximately 50 percent of normal individuals. Therefore, a red-lens diplopia should not be disqualifying for flying unless it increases in amount in the field of action of an ocular muscle, indicating a muscle paresis which is the purpose of the test.

2. An individual can fuse a blue and red light most easily, a blue and white light with less ease, and a red and white light with least ease. This is possibly explained on the basis of retinal sensitivity to intensities of light, the blue threshold having greater similarity to the white than the red. Since the combination of red and blue lenses seems to be fused most easily, this might be the best method for diplopia testing.

3. Routine diplopia testing for muscle paresis is probably better done in the primary position at six meters rather than at 75 cm., since the normal variation for phorias is less in amount at distance than at near. The greater the phoria, the more difficult it is to fuse the colored glass. This is in agreement with Wolpaw who has shown that red-glass diplopia in the primary position is quite common in individuals with exophoria or convergence insufficiency when tested at 75 cm.

4. Breadth of fusion (vergences) showed no relationship to the existence of a red-lens diplopia in this series of cases.

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UNUSUAL ULTRAVIOLET-RAY BURN OF THE EYES*

ROCKO MICHAEL FASANELLA, M.D.
New Haven, Connecticut

A 32-year-old man, manager of a tile and floor covering company, was seen at 3:30 a.m. with a history of sudden, intense pain, and lacrimation which awakened him from a sound sleep at 1:00 a.m.

He gave a history of having substituted at an inn for a good friend who had to attend a wedding. Recent exposure to ultraviolet radiation of any form was denied. One week previously he had glanced for an instant at a welder and six days previously he had a two-minute exposure to ultraviolet to his neck following a shave in a New York barber shop, "to prevent pimples that I get following a shave."

Exposure to excessive sunlight during the day, which had been a hot, bright, summer one, was also denied.

The patient was guided into the examining room by his wife. Examination showed an apprehensive individual with such severe photophobia that visual acuity could not be obtained.

After instillation of pontocaine (0.5 percent) anesthesia, an acuity of 20/30 plus in both eyes was obtained. There were small pupils and injection of the bulbar conjunctiva. A diffuse superficial punctate staining of the cornea of both eyes with two-percent aqueous mercurochrome was noted with the slitlamp. There was pain to palpation over both trochlear areas.

The patient was temporarily given holocaine and epinephrine ointment and a prescription for equal parts of 0.25-percent pontocaine and 0.25-percent neosynephrine (ophthalmic) to be obtained later that morning.

When seen the following day, the patient was symptom-free, with essentially negative eye findings. He had, in the meanwhile, uncovered the information that the inn owner and two other employees had off and on complained of milder but similar eye troubles. A fourth regular employee had never been troubled with these complaints. The latter was the only one who wore ordinary glasses for constant use.

The source of ultraviolet exposure was a modern, "germ-killing" ultraviolet lamp adjacent to the sink where beer and cocktail glasses were permitted to dry. My patient had been working over the sink for four hours that day.

DISCUSSION

This case shares in common the etiology of "An unexpected ultraviolet-ray burn received in an unusual manner," by Downing, Baird, and Wyman,* who reported a man aged 67 years, was admitted to the Boston City Hospital skin clinic with a complaint of intense burning over the lower back and upper buttocks.

In their case, the patient had stopped at a restroom in a bus station where ultraviolet-ray germicidal tubes were used for restroom sanitation. He sat on the toilet approximately 10 or 12 minutes, at the end of which time he noted burning and tingling of his back. That night he had considerable discomfort and was unable to lie on his back.

Physical examination of the lower back and upper buttocks on the following day showed a skin that was brilliantly red, with a few scattered vesicles. On palpation the skin was very tender. It was suggested that the company manufacturing the lights devise some method of disconnecting the current while the toilet was in use.

In my case of ultraviolet burns of the eyes it is interesting to note the lack of knowledge of the inn employees of the source of their ocular complaints. The protection afforded to the employee who wore ordinary glasses is logical and can be offered as one suggestion for eye protection. A second suggestion is a shield or shields between the sink and the sterilizing shelf.

* From the Section of Ophthalmology, Department of Surgery, Yale University School of Medicine.

* New England J. Med., **244**:876 (June) 1951.

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

SECTION ON OPHTHALMOLOGY

December 20, 1951

WILFRED E. FRY, M.D., *chairman*

ANNULAR MELANOMA OF THE IRIS AND CILIARY BODY

DR. PAUL A. ROCKWELL (by invitation) reported the case of a 49-year-old white man who presented himself with a retinal detachment. A peripheral disinsertion was described, and successful transscleral diathermy performed. The retina remained flat for 10 months and then recurrence of the detachment and signs of raised intraocular pressure developed. At this time a tumor was apparent, growing from behind the iris. The eye was enucleated and an annular malignant melanoma was found to occupy the entire ciliary body.

The author suggests that the reported cases do not permit accurate dividing of annular tumors into those of the iris and those of the ciliary body. He wishes to classify them simply as annular malignant melanoma of the iris and ciliary body. He has collected 31 cases from the literature.

Discussion. Dr. A. Valdes-Dapena (by invitation) said that he had the opportunity of seeing the specimen to which this presentation refers. Because of the clinical diagnosis of probable tumor of ciliary body and because transillumination failed to show anything in the posterior hemisphere of the eye, the latter was cut with a frontal section.

This revealed a large retinal detachment involving approximately one half of the eye and a grayish tumor mass disposed in a ring-like fashion around the iris. It was not uniform, but presented a nodular appearance.

The retinal detachment and the general features of the tumor as seen in a Kodachrome photograph were presented. Sections were taken and embedded in paraffin.

The microscopic picture was that of a very cellular, quite uniform tumor made up of plump epithelial-like cells endowed with moderately large rather vesicular nuclei with prominent nucleoli. There were scattered clumps of pigment, some of it intercellular and some of it within the cells themselves. There was no infiltration of the iris in any of the sections.

The histologic appearance was that generally referred to as epithelioid melanoma. Silver stains were made by the Laidlaw technique and a very fine delicate network of reticulin fibers was disclosed. This is in contrast to the rather heavy framework of reticulin which is found in the spindle-cell type of melanoma.

The importance of the histologic structure of this tumor refers mainly to its prognosis. The average melanoma of the eye is of the spindle-cell type and carries a fairly good prognosis. In the ciliary body it is more common than elsewhere in the eye to find an epithelioid variant of this pigmented tumor. It conveys a poor prognosis.

Reference has been made in the literature to the correlation between the amount of reticulin fibers and the relative prognosis. Again in this case the scantiness of the silver-staining network portends an unfavorable outcome.

A word might be said on the subject of the term melanosarcoma as against that of melanocarcinoma. It is most widely accepted that melanoblasts are of ectodermal derivation. From that standpoint a term such as melanocarcinoma would appear more proper, regardless of the structure of the tumor. However, no such adherence to embryologic concepts is observed in every day descriptive pathology.

We are more likely to employ the term sarcoma in reference to tumors that morphologically correspond to the connective-tissue group and carcinoma to those that put on an epithelial appearance. By this token the subdivision of the melanomas into carcinomas and sarcomas might be justified. In order to avoid controversy, as I believe Dr. Rockwell mentioned, the simple term melanoma is employed, as a rule.

To return to the case in point, the findings in our laboratory tend to show that the tumor we are discussing arose in the base of the ciliary body and remained confined in its growth to that organ, thus producing a so-called ring melanoma. The clinical impression and Dr. Rockwell's contentions are thus sustained.

Dr. George F. J. Kelly: Is the reduced intraocular pressure in these cases due to partial destruction of the ciliary body?

Dr. Paul A. Rockwell: Dr. Dapena and Dr. Spaeth have added considerably to this presentation. I thank them for it. Dr. Kelly suggests that the involvement of the ciliary body could explain the low tension in this case. This is certainly a reasonable suggestion. As to Dr. Fry's question about differentiating this condition from scleritis, I can only say that, if the problem puzzled Dr. Fry, it would certainly puzzle me.

Ocular manifestations in leprosy

Dr. P. J. Kennedy: The leprosarium of the Bahamas is on the same island as the city of Nassau, situated about seven miles from it. There are 20 patients. An attending physician visits twice a week. There are no nurses. At the time of our visit, all of the patients were ambulatory except one who had a severe case of exfoliative dermatitis. The others all seemed to be in fairly good physical condition.

The ages of the patients ranged from 23 to 63 years. There were 17 males and three females; 17 white persons and three Negroes. The ocular findings in 39 eyes were as follows: one—enucleated; nine—choroiditis; 12—keratoconjunctivitis; five—early iris changes; two—advanced iritis with synechias and secondary cataract; two—phthisis; five—mature cataracts; six—immature cataracts; six—normal.

Six of these patients would be greatly benefited by cataract surgery, but there are no facilities available for surgery and no one qualified to operate. Most of the eye surgery of the Bahamas is done in the United States which is one hour away by air.

About 85 percent of the eyes are involved to some degree. Based on the work and reports of Elliott and his co-workers, it is safe to assume that this figure would reach 100 percent if the disease were present longer. It is unfortunate that adequate eye care is not available for these patients.

Discussion. Dr. Joseph V. Klauder: I need not tell you that leprosy is one of the oldest diseases known to man. It existed in the Confucian era and in Biblical times. Interestingly, of all systemic diseases leprosy has the highest incidence of ocular manifestation. This incidence as stated by different writers ranges as high as 90 percent. Moreover, if the patient lives long enough and the disease is unchecked by treatment, industrial blindness ensues in a high percentage of patients. In a consistent manner, 17 of 20 lepers that Dr. Kennedy and I studied had ocular involvement. The estimated number of lepers in the world today is from three to five million, and in the United States the estimate is from one to two thousand.

In the United States cases arise in Florida, Louisiana, Texas, and southern California. About 400 patients are under treatment at the National Leprosarium, Carville, Louisiana. In consideration of airplane travel the United States is not remote from heavily infected localities—certain parts of the West Indies, Central and South America. Moreover, thousands of Americans were exposed to leprosy in World War II, and may still be exposed today since the southern part of Korea is an endemic region.

The advent of slitlamp microscopy has focused attention on the importance of ophthalmologic examination in early diagnosis of the disease. Leprosy then is of particular interest to the ophthalmologist.

The causative agent—Mycobacterium leprae—has never been cultivated or successfully inoculated into lower animals. It is not known whether the organism lives outside the human tissues. A similar acid-fast bacilli organism—murine leprosy bacilli—causes rat leprosy. Although this organism successfully inoculates laboratory animals, it likewise has not been grown in vitro. It is generally conceded that rat leprosy is not the same as that which affects the human being.

The mode of transmission of leprosy disease is not definitely known. It is believed that the route is the skin and/or nasal mucous membranes. Close association over a long period of time with a patient who has the lepromatous (open) form of the disease is apparently required. Infants and children are highly susceptible and are removed from the environment of the disease. The estimated risk of children under the age of 15 years in the household of a lepromatous case is 1 in 70, that of adults, aged 15 years or over, is 1 in 600.

The disease is more likely to be contracted in childhood than in adult life. Data suggest that transmission to a marital partner is unusual. Attendants in leprosaria rarely contract the disease. More recently such attendants are given a preëmployment lepromin test to determine their immunity to the infection.

The incubation period may be as long as 10 or even 20 years, and as short as a few weeks or months. The average incubation period is two to three years.

New concepts of leprosy pertain particularly to classification, laboratory tests, and treatment. The older classification—cutaneous, neural, and mixed types—is no longer in vogue. The newer classification is a correlation of clinical manifestations with histopathologic changes, test of immunologic response, and bacteriologic findings. It embraces the following two forms—lepromatous or nodular and tuberculoid.

In between these two basic types of leprosy there are other forms in which there has been much controversy regarding their designation, classification, and meaning. This is designated as simple inflammatory, mixed, indeterminate, or nonspecific.

Lepromatous leprosy comprises nodules and lepromas which are infiltrated plaques affecting different regions of the skin and mucous membranes, loss of eyebrows, rhinitis, fever, and peripheral pains. This form is mutilating, causing destruction of the nose and extremities. Histologically there is a granulomatous infiltration and changes suggestive of poor defensive mechanism to the invading organism. The bacilli are demonstrated in the skin and in and beween nerve fibers. The lepromin test (Mitsuda test—an interdermal injection of antigen made from tissue rich in M. leprae) is invariably negative, evidencing little or no immunologic reaction to the infection. This form is the most serious and severe form of the disease.

The tuberculoid form is the neural type of the older classification and is presented as small nodules and papules and as flat, round, or oval plaques. The palpable nerves are enlarged, fever is absent, and atrophy of the hands occurs. Histologically there is a patchy sarcoid—like areas of infiltration around hair follicles, blood vessels, and glands. The changes suggest a good defense of the tissues to the invading organism. The bacilli are difficult to find. The Mitsuda test is invariably positive. This form of leprosy is benign and spontaneous arrest of the disease may occur.

The simple inflammatory form is characterized by macular lesions and pinkish or dyschromatic areas of anesthesia. Histologically there is lymphocytic infiltration around blood vessels, glands, and hair follicles. No or only occasional bacilli are present. The Mitsuda test is positive in 40 to 50 percent and negative in 50 to 60 percent.

deBarros, Vale, Pinkerton, Elliott, and others have reported ocular manifestations in all types of the disease, but generally more frequent in the frank lepromatous forms.

There is a peculiar susceptibility of neural tissue in leprosy. Pathologists have emphasized that to the histopathologist, all leprosy is neural leprosy. The cutaneous nerves related to the skin lesions are involved in all types of leprosy. It is not surprising therefore that nerve filaments in the cornea are not exempt.

In therapy certain derivatives of chaulmoogra oils are considerably less employed. The most important single therapeutic advance occurred with the introduction of the sulfone drugs. The ones most widely used are glucosulfone sodium (promin), sulfoxone sodium (diasone), and sulfetrone. The most satisfactory method of administration is the oral route, although some of the sulfone drugs can be given intramuscularly or intravenously.

A deficiency of sulfone treatment is the very slow rate at which the bacilli are eliminated in the skin and nerves. Prolonged treatment over a period of years may be required to cause complete disappearance of the bacilli and arrest of the disease. Dihydrostreptomycin has now replaced streptomycin and is used as an adjunct to sulfone therapy, especially when improvement in skin and mucous membrane lesions has become stationary. This therapy is also useful to treat the so-called lepra reaction.

Other drugs still in the experimental state are being investigated. Mention may be made of aureomycin, para-amino-salicylic acid, and more especially of amithiozone (tibione) (4-acetylaminobenzalthiasemicarbazone). The latter drug appears to be the most promising and in some quarters is used in conjunction with the sulfones.

Sulfone therapy is valuable in prevention of ocular manifestations when employed in the early stage of leprosy. When ocular involvements are far advanced, the process may advance despite sulfone therapy.

In summary the earliest ocular changes in leprosy (Pillat, Vale, Elliott, and others) is a beaded appearance of the fine filaments of the corneal nerve and many glomeruli or tufts of capillaries at the equator of the limbus and laterally. This perineural infiltration is soon followed by a nebulous keratitis. Nodular iritis with many synechia and lenticular changes is a frequent occurrence, as is, later in the course of the disease, a gross interstitial keratitis.

Discussion. Dr. R. D. Harley: The observations on ocular leprosy presented to you by the essayist reflect many hours of hard work and infinite technical knowledge.

The subject was of particular interest to me since I was permitted the unique opportunity of studying the eye complications of this disease for approximately four and one-half years while stationed at an army hospital in the tropics.

The ocular complications of leprosy are extremely high and amounted to about 90 percent in our series. Most of these patients were moderately advanced mixed cases; that is, a combination of the nodular and anesthetic type. Only 25 percent of the patients were of the purely anesthetic type. Pinkerton in Hawaii and Prendergast at Carville found the same percentage of involvement in their series. The eye complications, although not generally known, offer the greatest single threat to these patients, since individuals rarely die of the disease.

This fact was brought out in the survey which disclosed that 13 percent were blind in one or both eyes while an additional 41 percent have vision of 20/200 or less in one or both eyes, not correctible. Thus 54 percent of all patients had severe visual loss amounting to 20/200 or less in one or both eyes which in most instances was attributable to the disease.

The cornea appeared to be the most vulnerable ocular tissue to Hansen's disease—some form of corneal involvement was found in 58 percent of all patients. They have been classified as three principal lesions: (1)

Superficial punctate keratitis, (2) deep interstitial keratitis, and (3) pannus or infected granulomatous keratopathy which extends in from the limbus.

The most commonly observed corneal lesion was superficial keratitis which occurred in 34 percent of all cases. It usually began as a light, milky, superficial haze punctuated by tiny white dots at the superior or superior temporal limbus. The lower margin of the lesion was well delineated by a wavy line which often ended just above the pupillary border of the cornea. The involved area gave the impression of a finely pulverized "chalk" or "flour" dust as has been described by others. Many of these patients had normal vision and were unaware of any difficulty. The lesions were easily visible by slitlamp microscopy when the upper lid was elevated.

Histologic examinations of these corneas showed aggregations of round cells and polymorphonuclears beneath Bowman's membrane leaving the epithelium intact. Lepra cells were identified.

"Beading" of the corneal nerves seen under high magnification is an interesting sight and easily overlooked.

Absolute corneal anesthesia was observed in only two far-advanced cases.

When a small wisp of cotton is used, relative anesthesia was found in a small number of cases. When a heavier stimulus is used, a pain sensation was easily demonstrated. It appeared that corneal anesthesia was not nearly so common as it was once thought to be.

Next to the cornea, the iris is the structure most frequently involved. Iritis and/or its sequelae was recognized in 50 percent of the patients. It was usually bilateral, and occurred in recurrent attacks. The most fascinating slitlamp picture of the iris was the tiny lepromas seen in over 26 percent of the cases. They were grayish yellow, pedunculated, pinpoint-sized bodies usually seen about the pupillary rim. They often appeared in clusters and occasionally were seen near the limbus. Sometimes larger lepromas were seen.

It appeared that the superficial punctate keratitis and the tiny lepromas of the iris were so characteristic of the disease as to be considered diagnostic.

Conjunctival smears were done in 100 cases but lepra bacilli were found in only eight slides. Conjunctival lesions are not common, and this structure seems relatively immune. It was believed that the bacilli might have entered the conjunctival sac as a contaminant from the skin.

Hansen's disease involves the region of the eyebrows and eyelids with relative frequency. Hypertrophy of the skin with subcutaneous nodules and loss of eyebrows or lashes accentuates the features and folds so that a "leonine facies" develops. Loss of lashes appears to be a late development.

The posterior segment of the eye seemed to be rarely involved in this series. Leprosy characteristically involves the anterior segment of the eye.

Promin was begun at the colony in 1945 with spectacular results. The open skin lesions healed, nodules flattened, and recurrent iritis was definitely reduced.

Marriage between patients was permitted. Infants resulting from this union were removed at birth. There have been no known cases of the disease occurring in the children of these patients in over 30 years.

Discussion. Dr. Edmund B. Spaeth: I also was an attending ophthalmologist to a leprosarium, in the Canal Zone for about 10 months, and have one comment to add to Dr. Harley's extensive experience. The lepra bacillus can be found in the nasal mucosa and in the lacrimal sacs in practically all cases of this disease. It is undoubtedly through the lacrimal canaliculi that these bacilli gain admission to the conjunctival sacs. This mode of entrance has been suggested, however, before.

Dr. R. D. Harley: The remarks of Dr. Spaeth concerning involvement of the nasal mucous membrane in Hansen's disease re-

minds me that the tear sac was omitted in the discussion.

A purulent dacryocystitis was noted in five percent of the cases. Stricture or other occlusive phenomena of the nasolacrimal duct which lead to tear-sac infection is probably always secondary to advanced nasal disease. Erosion of the lacrimal bone was encountered on one occasion when a dacryocystectomy was being performed. Dacryocystorhinostomy is not indicated, due to the extremely diseased nasal mucous membrane in leprosy.

Patients with Hansen's disease withstand surgery better than one might expect. Cataract extraction is always complicated by the adhesions to the iris. Corneal transplantations were performed, but all of the grafts turned opaque after two or three weeks.

CORTISONE AND EXPERIMENTIAL CORNEAL BURNS

DR. IRVING H. LEOPOLD, and DR. FLORIAN R. MAYLATH (with the technical assistance of John Sawyer) made a study of topically applied cortisone-acetate suspension. The agent penetrates readily into the anterior chamber of normal rabbit eyes. Continuous daily therapy allows a maximum level to be reached which is not exceeded by continued treatment. Cessation of therapy is followed by a rapid drop in the anterior chamber concentration of cortisone within a subsequent 24 hours.

Topical therapy with cortisone-acetate suspension allows detectable but not high levels of cortisone to be found in the vitreous humor. Cortisone disappears from the aqueous humor within 24 hours after the cessation of therapy but not quite so rapidly from the vitreous humor.

Subconjunctivally administered cortisone produced high concentrations in the aqueous humor, more rapidly than following drop therapy. Vitreous concentrations following this route of administration were higher than after topical administration. Within 24 hours after cessation of subconjunctival administration the cortisone levels rapidly diminished in the anterior chamber and somewhat more slowly from the posterior chamber.

Systemically administered cortisone penetrated readily into the aqueous and vitreous humor. The levels obtained in the aqueous humor were higher than in the vitreons humor. The rate of penetration into the aqueous humor was slower following systemic administration than after subconjunctival administration. The rate of appearance of corticoid materials after intramuscular and intravenous corticotropin was slower and less in amount than after intramuscular cortisone.

All methods of therapy—topical, subconjunctival, and systemic—produced a less dense scar in the thermal, acid, and alkali burns of the cornea as compared to the untreated control eyes.

Cortisone therapy reduced the extent of cornea vascularization following severe alkali burns.

Discussion. Dr. Irving H. Leopold: There are a few observations which Dr. Maylath did not mention in his excellent presentation. It should be mentioned that this method is not specific for cortisone. There are other substances which will also give a reaction with phenylhydrazine. At the present time studies are underway to identify these other positive reacting substances. The method under investigation is the filter-paper chromatography technique.

Repeated paracentesis of the anterior chamber produces a definite rise in the concentration of the color-producing material in the secondary aqueous humor. This may indicate that there is an increase in these corticoid materials in the secondary aqueous humor. However, further analyses will be necessary to confirm this observation. If this is borne out by further investigation, it would suggest that some of the beneficial effect of paracentesis in uveitis and keratitis is not only due to the influx of immune bodies in the secondary aqueous but also to the influx of these corticoid materials.

M. Luther Kauffman,
Clerk.

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OPHTHALMIC LABORATORIANS

Now that the summer holidays are over we can start thinking once more about ophthalmology and its many unsolved problems. To be sure some of us have performed informal and not strictly scientifically controlled piscatorial experiments, during which time, alas, our tempers may not have been always properly controlled either. An ophthalmic friend, for example, spent many hours wondering why a fish's eye on the end of a hook had such an attraction for another fish, and being somewhat of a philosopher amused himself and his cronies by playing with the obvious moral to be drawn. The Compleat Angler has a verse in which he says:

> "Here are no false entrapping baits
> To hasten too, too hasty Fates,
> Unless it be
> The fond credulity
> Of silly fish, which worldling like, still look
> Upon the bait, but never on the hook."

But it is time to put away these thoughts and pleasures and turn to more serious

things, for autumn is upon us and there is work to do, investigations to make, students to be trained, meetings to attend, and scientific papers to prepare.

Those investigators fortunately placed in a well-equipped laboratory and competently trained in experimental techniques, are probably by this time completely absorbed in their work. Although few in number, we have learned to trust the integrity of their answers to many problems and have almost an inherent right to expect new and sometimes startling things at regular intervals from them. To be sure, some of these results do not have an obvious clinical bearing, but basic facts that are apparently irrelevant have a way of accumulating and in time fall in place like pieces of a jigsaw puzzle to give us a clear and significant picture.

The language these laboratorians speak or write is often beyond our immediate comprehension and one harbors, quite unjustly, the suspicion that even they themselves do not entirely understand each other very well.

They are generally modest fellows, too, who speak softly, a point that some of us loud and self-assured clinicians could well take under advisement. In formal scientific meeting, they are in the habit of making their points, asking their questions, or giving their answers in dulcet undertones back and forth in their own select circle made up of chairman (erudite fellow), essayist (on the spot), and the discussor (the fly in the ointment).

They cover the blackboard and the screen with the weird signs and symbols of their witchcraft, often lowering their soft voices just at the crucial point where the ordinary chap is almost on the beam with them. Their lantern slides consist of figures of staggering lines, dots, crosses, arrows, blocks, curves, Greek and presumably Armenian cabalistic letters, all probably of great significance to ophthalmology but usually leaving even the most experienced clinician far behind them in his mental digestive processes.

We, who are of far lesser stature, are lost in admiration over the mysteries that they reveal and are apt to read more into their work than they do themselves. Obscure and mysterious things have a peculiar psychologic fascination that conveys often more significance than is justified.

We have learned to love their signs and symbols, some of which are old friends by now, although we for the most part still don't know what they mean. It is, however, not the fault of our friends, the researchers, bless them. They are an unusually fine lot of chaps, not given to beating their rats.

We appreciate the ingenuity and artistry of an inspired bit of laboratory work, just as we appreciate the subtle intricacies of a fine symphony. We get an esthetic thrill over the beautiful precision of work based on data painfully gathered and are filled with awe.

Frequently we lose the thread of the discourse, the gist of the argument, the meat in the pie, and our minds are apt to wander. "What happens," we say to ourselves, "to the wives and laddies of these chaps who are busy days and nights for weeks on end measuring the light threshold, for example, of the end of a rod in the retina of the cat, or studying every hour on the hour the eyegrounds of rats that have been given some outlandish drug whose name begins here and ends way over there? Does the wife send the oldest child through the swinging doors of the lab to beg his father to come home with him now? Or does she go herself at the cocktail hour to enjoy a few minutes with her mate? And do they drink martinis made six to one, with extra laboratory distillation and measured scientifically into beakers with graduated pipettes?"

Thus our thoughts wander until we are suddenly brought back by the words of the speaker whose conclusions, while relevant to the cat or rat, have no apparent resemblance to any person living or dead—a point which he makes very clear. Does he perhaps share with the author and the playwright the fear of a long and expensive lawsuit with all

the penalties and costs pertaining thereunto?

We wistfully recall at this point the words of Isaak Walton "and upon all that are lovers of virtue; and dare trust in His providence; and be quiet; and go a-Angling. Study to be quiet."

Georg Bartisch II.

XVII INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

The XVII International Congress of Ophthalmology will take place at the Waldorf-Astoria Hotel in New York City the week beginning September 12, 1954, marking 97 years after the meeting of the I International Congress held in Brussels in 1857. This will be the second time in 78 years that the congress has been held in the United States, the previous meeting being the V International Congress held in New York in 1876. The last meeting was held in London in 1950.

The meetings of the International Congress take place every four years except for interruptions caused by wars. They are conducted under the authority of the International Federation of Ophthalmological Societies, one of the oldest of all international organizations. The broad aim of the federation, which is composed of delegates sent by ophthalmological societies of many countries, is to promote the science of ophthalmology throughout the world.

The International Council, meeting recently in Oxford selected the following subjects to be discussed at the congress: "Primary glaucoma" and "Etiology of uveitis." There will be three main papers on each of these subjects.

Those on "Primary glaucoma" will be:

1. "Etiology and general considerations," Dr. Derrick Vail, Chicago, Illinois.

2. "Medical therapy," Dr. R. Thiel, Frankfurt a/Main, Germany.

3. "Surgical treatment," Dr. Gabriel Sourdille, of Nantes, France.

The three main papers on "Etiology of uveitis" will be:

1. "General considerations," Dr. Alan C. Woods, Baltimore, Maryland.

2. "Allergic factors," Dr. Norman Ashton, London, England.

3. "Virus factors," Dr. Vittoriano Cavara, Rome, Italy.

Those who wish to present dissertations or to participate in discussions should communicate with the secretary general, Dr. William L. Benedict, Rochester, Minnesota. English, French, and Spanish are the official languages of the congress but any language may be used. Communications should not be longer than 2,500 words and will be read in abridged form. On submission each paper should be accompanied by a résumé of about 250 words in each of the three official languages. Papers published previous to the meeting of the congress are not acceptable. The Publication Committee reserves the right of rejection.

OBITUARY

RICHARD GORDON SCOBEE (1914-1952)

Richard Gordon Scobee died suddenly the night of Sunday, June 22, 1952, as he was relaxing over his stamp collection. The loss to his friends and to ophthalmology is overwhelming for in the 10 years between the completion of his training and his death he revolutionized the teaching and practice of strabismus correction. He was so full of life and fun, and such an inspiring teacher, that all who knew him cannot fail to remember him with warmth and admiration. Many times those of us who were close to him professionally received a powerful impulse to do better. We learned to gather and analyze data, to draw conclusions from the data, and to act decisively on them. He was the spark plug of our department and, without him, things will be dull indeed for many

of us. Perhaps his most indicative professional characteristic was his willingness to change his mind when new information showed that his former ideas were incomplete or wrong.

In 10 years, Dr. Scobee produced a bibliography of 70 items, including two textbooks.

Richard Gordon Scobee

He lived to see the second edition of his textbook, *The Oculorotary Muscles*, published. He frequently made a diagnosis in one or two minutes, using a flashlight and his hand, in the presence of other experts in muscle anomalies who had arrived at the same diagnosis only after long and laborious study with other methods. He taught dozens of ophthalmologists his simplified method of muscle analysis and it has been widely accepted throughout the country as a valid and accurate clinical method. His great achievement was removing the mystery from squint and in teaching other physicians how to follow his methods.

Dr. Scobee was born in Sherman, Texas, on November 14, 1914. He put himself through college (Rice Institute, Houston, A.B., 1935) and medical school (the University of Texas, M.D., 1939) and received his early ophthalmologic experience as office assistant to E. W. Griffey, M.D., during the summers of 1935, 1936, and 1937. He interned at the Methodist Hospital, Fort Worth (1939-1940), and received his ophthalmologic training at Washington University School of Medicine and Barnes Hospital, St. Louis (1940-1942). He was interested in the study of squint from the beginning of his ophthalmologic career.

On his return from his military service in 1945, he became assistant in ophthalmology in the Department of Ophthalmology at Washington University, in charge of postgraduate teaching. He was an assistant professor by 1948, and remained in charge of postgraduate teaching until his death.

Dr. Scobee was in charge of the Ophthalmology Section of the School of Aviation Medicine at Randolph Field during his military service from January, 1944, to September, 1945. During this period, he became increasingly interested in heterophoria and contributed much to the study of the effects of heterophoria on pilot efficiency. He taught and studied tirelessly and introduced hundreds of military physicians to ophthalmology during this period. He was consistently voted the best teacher in the School of Aviation Medicine by several successive classes at Randolph Field.

On returning from military service, he reëstablished the course of postgraduate training in the basic sciences in ophthalmology at Washington University and became its best teacher, its organizer, its inspiration, and its general ferment. At the same time, he plunged deeply into the study of anomalies of the oculorotary muscles, amassed an astonishing number of accurately studied cases, and re-organized the special "muscle

service" which was, in the eyes of the students and the resident staff, the best feature of the department's postgraduate teaching.

Dr. Scobee was one of the earliest members of the NRC-Vision Committee of the Armed Forces (the former Army-Navy NRC-Vision Committee) and, after a one-year term as chairman of this committee, he remained a member of its executive council until his death. He never missed a meeting of this committee, from its inception in 1944 to the most recent meeting in April, 1952. His common sense and impatience with pompousness kept pulling the vision committee back to "vision" whenever it went too far astray in psychophysical statistics, and he gave a great deal of himself to these meetings. His first coronary occlusion occurred in April, 1951, in Washington while attending a meeting of this vision committee.

It was on trips to these meetings that I came to regard Dr. Scobee as a close friend and learned that he was completely intellectually honest, as well as a man endowed with a remarkable sense of humor and a remarkable ability to organize. He was addicted to detective stories when time permitted and, when nothing else was at hand, he was humming a tune or telling a story.

The first edition of Dr. Scobee's textbook, *The Oculorotary Muscles*, appeared in 1947. It was rewritten for the second edition which was published a few weeks before his death. This textbook and his teaching in the classroom were his greatest contributions to ophthalmology. He made simple what generations of ophthalmologists had made difficult, and brought about a great change in the methods of diagnosis and treatment of heterophoria and heterotropia.

In his last three years, he became increasingly interested in orthoptics and re-established the Orthoptic Clinic at Washington University. He became chairman of the committee on instruction of the American Orthoptic Council, and the basic orthoptic course was transferred to St. Louis this year. He had the course completely organized and ready to go, but died eight days before it began. Typically, Dr. Scobee had just completed teaching a personal, intensive muscle course the day before his death, and was ready to begin the orthoptic course when his career ended.

In short, Richard Scobee managed all aspects of a physicians' life with great success. As a practitioner, as a teacher, as a departmental organizer he was professionally far above the rest of us, and in his few years made heterophoria and heterotropia far easier for us to understand. His patients, mostly children, loved his sense of humor and kindness. As a colleague and friend, he inspired one at all times to accomplish more. As a research man, he made sense out of the many tests for heterophoria.

In coöperation with the National Society for the Prevention of Blindness, he and his colleagues organized and conducted a study of 1,500 school children to determine which method of visual testing in the schools would be the most reliable visual screening test. These data are to be published posthumously. Further, he analyzed more than 2,300 cases of heterophoria and heterotropia, and discovered some previously unknown facts. Still further, he conducted numerous studies for the NRC-Vision Committee and the Office of Naval Research. As a family man, he was a devoted father and husband, and in spite of all his other activities, was able to be at home more than most physicians.

Dr. Scobee packed into his 10 professional years more than most physicians accomplish in 50, and his loss to all who knew him and worked with him is truly a great one. He is survived by his wife, who was head nurse in the Eye Clinic when he was house officer, their two children, and his parents.

A list of the professional societies of which Dr. Scobee was a member includes: diplomate, American Board of Ophthalmology; fellow, American Academy of Ophthalmology and Otolaryngology; fellow, American College of Surgeons; fellow, American Medical Association; American Committee

on Optics and Visual Physiology, American Orthoptic Council, Committee on Human Resources of the Research and Development Board, Association for Research in Ophthalmology, Council on Physical Devices of the American Medical Association, Vision Committee of the American Standards Association, St. Louis Ophthalmic Society, Missouri State Medical Association, St. Louis County Medical Society, Vision Committee of the Armed Forces Research Council, and consultant in ophthalmology, Office of Naval Research, Department of the Navy.

Philip T. Shahan.

CORRESPONDENCE

Use of chloral hydrate in cataract surgery

Editor,
American Journal of Ophthalmology:

As pharmaceutical knowledge increases, new alkaloids, synthetic drugs, synergists, or combinations of drugs are being used to obtain definite results for accurate assistance in very special forms of surgery. This is certainly true of ophthalmology and, with the advent of the intracapsular operation, more and more attention has been given to basal sedation, analgesia, and akinesia in order to assist in developing consistent perfection in operative technique.

It is not my intention to dwell on these important topics as they have been well covered by other writers. I wish, however, to call to your attention the use of chloral hydrate as a safe synergistic drug used in combination with the barbiturates, demerol or curare. For those who have had little experience in the administration of curare, chloral hydrate will be, in my opinion, a most welcome addition to their medical armamentarium. This drug cannot replace curare by any means but it can be of general assistance.

In my medical school days chloral hydrate was looked upon as a dangerous cardiac depressant, but there is no question that this is a fallacy, and it is my prediction that this drug will be used more and more. It has been impossible for me to discover any possible contraindication for its use.

The drug is detoxified in the liver and eliminated by the kidney, but impaired function of these organs does not prohibit its use. It is the oldest of the hypnotic agents having been introduced by Liebrich in 1869. The drug has been used in medicine for nearly a century and clinical experience shows that it is the safest of all sedatives.

In ophthalmology, chloral hydrate has been administered in the form of an enema. This mode of administration does not seem practical to me, since it is impossible to give an exact dosage. I know of a case in which the chloral hydrate for an enema was compounded in grams instead of grains. Even after this tremendous dosage, with the exception of about three days' sleep, the patient recovered with no ill effects.

When chloral hydrate is used in combination with other barbiturates, in order to get perfect basal analgesia some form of opium or a drug having some of the medical attributes of opium usually has to be used. This is also true if we wish to establish perfect analgesia when curare is used, for curare is concerned principally with akinesia.

Morphine, because it stimulates the emetic center in more than a quarter of the patients to whom it is given, with ensuing nausea and vomiting, is certainly contraindicated as an agent in basal analgesia. The synthetic narcotic, demerol, is to date the best drug for this purpose.

Demerol is an atropine analogue. After intensive pharmacologic studies in animals, its morphinelike properties were discovered. Subsequently, they were confirmed in man. Demerol can be designated "little morphine," that is, it has all the principal properties of morphine but to a lesser degree. It does stimulate the emetic center which is of interest to the ophthalmologist; it rarely depresses the respiratory or pupillary centers, and the cough center is unaltered.

The discussion now comes back to chloral

hydrate. In the first place this drug has a wide margin of safety. As a synergist, it works well in combination with other medicaments during the surgical treatment of cataract. In my opinion, the best and safest way to administer the chloral is in a rectal capsule.

This method is accurate and foolproof.

Knowing that the Fellows Pharmaceutical Manufacturing Company, Inc. was especially interested in chloral hydrate, I consulted Dr. Arthur E. Meyer, director of research, and he had made for me rectal capsules containing 20 gr. of chloral hydrate which a number of my colleagues and I have used with great success.

These capsules are moistened and inserted in the rectum one and one-half hours before operation. The capsule is absorbed in a matter of minutes. It is well-known that the chief disadvantages of chloral hydrate are its taste and gastric irritation. However, since the preparations of chloral hydrate dispensed by the Fellows Company are dissolved in a selected combination of glycerol esters and enclosed in a soft gelatin capsule, these objectional features are entirely eliminated, even when long-continued use is necessary.

I have used chloral hydrate rectal suppositories (20 gr.) in combination with other medication in squint operations and enucleations. In my opinion they give added sedative and analgesic effects. I venture to predict that in the years to come our internist colleagues will dust off an old bottle, take it from the shelf, and become more familiar with one of the safest, oldest, and most reliable sedative and hypnotic agents.

(Signed) William Brown Doherty,
New York, New York.

BOOK REVIEWS

B FOR MEDICAL WRITING. By E. P. Jordan, M.D., and Willard C. Sheppard. Philadelphia, W. B. Saunders Company, 1952. 103 pages, 26 figures, appendices, index. Price: $2.50.

The subtitle of this book is "A useful guide to principles and practice of effective scientific writing and illustrations." The opening verse is by Alexander Pope who said:

"True ease in writing comes from art, not chance,
As those move easiest who have learned to dance."

The editor of any medical journal will welcome this brief but splendid guide for use by his contributors and the secretary of any medical society will wish his organization could send a copy to each essayist.

There are 20 short chapters covering the preliminaries, the first draft, the first revision, the second revision (there is a delightful paragraph here on obscure writing or "puddery"), the third revision, the beginning and the end, special problems, making the index, illustrations, and statistics employed in medical papers (which uncovers the mystery and slight of hand hitherto awesome).

There is great need for this book. Every author, potential writer, or would-be author of a medical paper should own and dog-ear a copy. It is worth four times the price to all concerned.

Derrick Vail.

CURRENT THERAPY 1952. Edited by Howard F. Conn, assisted by 12 consulting editors. Philadelphia, W. B. Saunders Company, 1952. 849 pages, index. Price: $11.00.

This excellent book continues the high standard set by its predecessors. Those who are familiar with the 1951 volume will need no encouragement to look into the present one. Those who are not familiar have missed a valuable tool in the understanding of

modern medical treatment. There are almost 400 contributors, whose names constitute a *Who's Who* in most of the branches of medicine.

The following diseases are discussed from a strictly therapeutic viewpoint: (1) The infectious diseases, (2) the respiratory diseases, (3) diseases of the cardiovascular system, (4) diseases of the blood and spleen, (5) diseases of the digestive system, (6) disorders of metabolism and nutrition, (7) diseases of the endocrine system, (8) diseases of the urogenital tract, (9) the venereal diseases, (10) the allergic diseases (of particular interest to ophthalmologists), (11) diseases of the skin, (12) diseases of the nervous system (of ophthalmic value), (13) diseases of the locomotor system, (14) obstetric and gynecologic conditions, (15) diseases due to physical and chemical agents, and (16) the appendices and index.

Every M.D. should have this reference book for handy and daily use, regardless of his specialty.

Derrick Vail.

ATTI DELLA SOCIETA OFTALMOLOGICA ITALIANA. *Rome, Arte della Stampa, 1951, v. XII.*

This volume contains the transactions of the regional ophthalmological societies of Italy during 1950.

Among the papers presented before the Ophthalmological Society of Lombardy is an extensive monograph by D. Sabbadini on the heredity of myopia. He presented 395 cases and gave extensive family trees. He comes to the conclusion that myopia is a hereditary disease, usually of the recessive type. He believes that mild and severe myopia are only two manifestations of the same disease and not two different entities.

M. Valerio showed for the first time the histologic picture of the so-called "Primary edema of the cornea." This condition occurs in middle-aged adults, mostly in women. In his case the author performed a perforating keratoplasty. The specimen revealed that the most striking changes were in Descemet's membrane which showed excrescences as in cornea guttata. The author believes therefore that this is a corneal dystrophy.

P. F. Ferraris de Gaspare stressed the importance of plotting the blindspot and angioscotomas during provocative tests for glaucoma. The author believes this to be an important diagnostic sign. The blindspot may be enlarged, even when the rise in tension is small. This test gives a clue as to how an increased intraocular pressure would affect the function of the eye.

M. Valerio discussed ocular manifestations of Still's disease. He stressed the importance of the triad, uveitis, band keratopathy, and complicated cataract, as diagnostic signs even when the manifestations of Still's disease are incomplete.

G. Margotta presented a case of orbital cellulitis following an extraction of an infected tooth. It was assumed that the infection spread as a thrombophlebitis. The young boy recovered on penicillin. L. E. Grancini spoke on the familial incidence of retinal detachment on the basis of his observation of this condition in two brothers.

A. Wirth discussed the clinical applications of electroretinography. The difficulties are now less of a technical nature but arise from variations in the interpretation.

Among the papers dealing with newer drugs was a report by J. Orsoni on the results of PAS therapy in ocular tuberculosis. The author had good results in seven cases. As PAS lowers the prothrombin level, vitamin K should be given simultaneously.

A. Bucalossi reported on the effect of sulfone in 31 patients with leprosy. The sulfones prevented ocular complications in 20 patients who were without them. It improved the ocular lesions of leprosy with the exception of interstitial keratitis and pannus. E. Leo found good results in seven patients with trachoma after oral chloramphenicol. The use of antihistamines in phlyctenular keratoconjunctivitis was discussed by R. Campos and S. Stagni. They found improve-

ment when pyribenzamine was used orally without any local treatment.

Among the surgical papers was a discussion by M. Valerio on the value of the injection of air into the anterior chamber, a procedure which the author recommends after cataract extraction.

G. Margotta described his method of excising a serous cyst of the iris. He used a trephine over the corneal periphery as Blaskovicz had originally suggested. This will prevent a rupture of the wall of the cyst.

D. Cattaneo described another method of suturing an iridodialysis. The author is obviously not familiar with the more elegant and simple method of treating such lesions with diathermy according to Safar (*Ophthalmologica*, **114**: 77, 1947).

Among the papers presented before the meeting of the Ophthalmological Society of Tuscany, Umbria, and Emilia was a report by G. DiLuca on the X-ray treatment of sympathetic ophthalmia. Good results were obtained in 27 cases in which the sympathizing eye had been enucleated. The author uses 120 to 150 r per dose. No other factors are given. A. Bucalossi showed an eight-months-old infant with five retinal tears in one eye without detachment. The tears were obviously metacystic. The child suffered from a postnatal encephalopathy with convulsions.

The second meeting of this Society in 1950 has already been reported in this JOURNAL (Am. J. Ophth., **35**: 280, 1952).

Many of the papers read before the meeting of the Ophthalmological Society of Sicily dealt with various aspects of trachoma.

Frederick C. Blodi.

A TECHNIQUE OF FITTING SPECTACLE FRAMES. Published by The Association of Dispensing Opticians, 1952. Paper bound, 20 pages, appendix. Price: 2s.6d.

This booklet, published by the British Association of Dispensing Opticians, is essentially an instruction manual for the neophyte in spectacle-frame fitting. Considerable attention is given to the basic principles of taking the necessary measurements, especially for the old-type saddle-bridge frame, now rather obsolete in the United States, but still used in Britain. There is insufficient detailed instruction on fitting the modern plastic frame.

While this is a good basic treatise it does not attempt to deal with the finer points of optical dispensing.

William A. Mann.

DIE TRÜBUNGSFORMEN DER MENSCHLICHEN LINSE (Opacities of Human Lens). By Prof. H. Sautter. Stuttgart, Georg Thieme (agents for the U.S.A., Grune and Stratton, Inc., New York), 1951. 140 pages, 191 illustrations, many in color, voluminous references, index. Price: D.M. 49.80.

This is a splendid little book, beautifully illustrated and printed on fine paper. The pictures are comparable to those published in Vogt's *Atlas of Slitlamp Microscopy* and can be reproduced on Kodachrome slides for lecture purposes.

The chapters include the biomicroscopy of the normal, diseased (opaque), and injured lens. All the known forms of lens opacities and the conditions that produce them are discussed in the light of our present knowledge.

It is not entirely necessary to read German to profit by possessing this small atlas. The pictures speak fluently for the subject. All those who are interested in cataract, and what ophthalmologist isn't, should own it.

Derrick Vail.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

5

DIAGNOSIS AND THERAPY

Goldmann, H. **A new slitlamp fluorometer.** Ophthalmologica **123**:277-280, April-May, 1952.

A new accessory to the Haag-Streit slitlamp, permitting insitu measurements of the fluorescein concentration in the anterior chamber of the intact human eye (after intravenous injection of the dye) is described and will be manufactured by Haag-Streit. Peter C. Kronfeld.

Hilding, A. C. **Instrument stand and drape support for eye surgery.** A.M.A. Arch. Ophth. **47**:639-640, May, 1952.

The author describes a device which consists of a small triangular table held in place by a rod, over which drapes are placed, which he has found useful, especially in cataract surgery. (2 figures)
R. W. Danielson.

Johnson, L. V. **Enucleation knife.** Tr. Am. Acad. Ophth. **56**:45-46, Jan.-Feb., 1952.

The author describes an enucleation knife which he feels has the following advantages: the sharper tip allows easier passage; and, more important, it allows the preservation of a scleral tab anterior to the muscle insertion. (1 photograph of the knife; and 2 pictures of the knife in use) Theodore M. Shapira.

La Motte, W. O., Tyner, G. S., and Scheie, H. G. **Treatment of retrolental fibroplasia with vitamin E, corticotropin (ACTH) and cortisone.** A.M.A. Arch. Ophth. **47**:556-569, May, 1952.

Observations were made during treatment of 17 premature infants with vitamin E, and 21 premature infants with corticotropin (ACTH) or cortisone. The authors conclude that neither method is effective. The results were almost identical; twelve patients in each group developed blindness in one or both eyes. There were several complications such as increase in body weight due to fluid retention, increased nutritional demands, increased susceptibility to infection, rounding of facial contours, and pustular dermatitis. R. W. Danielson.

Levitt, H. **The acorn implant.** A.M.A. Arch. Ophth. **47**:616-620, May, 1952.

The author describes an acorn-shaped buried orbital implant with tantalum

mesh on the large end. The technique of implantation with the mesh forward is described. This implant is similar to the ones used by Conrad Berens and A. Lemoine. (4 figures) R. W. Danielson.

Lijo Pavia, J., Arouh, J., and Lis, M. **General anesthesia in ophthalmology.** Rev. oto-neuro-oftal. **26**:9-12, Jan.-Feb., 1951.

The authors enumerate the advantages of a general anesthesia in ophthalmologic surgery. They emphasize that a trained anesthetist should give it.

Frederick C. Blodi.

Lijo Pavia, J., and Arouh, J. **Biologic therapy in ophthalmology.** Rev. oto-neuro-oftal. **26**:49-54, 73-85, 119-122, 1951.

The authors give a survey on the treatment of ocular affections with placental extracts. They find the evidence for a beneficial action contradictory and the initial enthusiasm has diminished. The authors are against using this therapy in cataracts, glaucomas or retinal detachment. It may be tried in disease in which the routine therapy does not succeed.

Frederick C. Blodi.

Magni, S. **Antiexudative and fibrinolytic action of cortisone in eye diseases.** Boll. d'ocul. **31**:147-156, March, 1952.

Four cases of plastic uveitis are described. The well-known favorable results were observed and explained. (references)

K. W. Ascher.

Mattis, R. D. **Paired-scissors design based on geometrical anatomy of the ideal corneal section.** A.M.A. Arch. Ophth. **47**: 621-624, May, 1952.

The author has attempted to design paired scissors which will duplicate as nearly as possible the knife section and which can be used with minimal trauma. After the original keratome incision, a single cut in one direction completes the section there, followed by one cut with matching scissors in the other direction. One must, of course, diligently watch the iris and preplaced sutures. (2 figures)

R. W. Danielson.

Mims, J. L., Jr. **Methyl cellulose solution for ophthalmic use.** A.M.A. Arch. Ophth. **46**:664-665, Dec., 1951.

Methyl cellulose solution is useful in replacing deficient lacrimal secretion, as a lubricant for prosthesis, as a protective medication for various pathological conditions of the cornea and after tonometry. The most satisfactory solution is one of 0.33 percent in a mixture of equal parts isotonic salt solution and Hind and Goyan's phosphate buffer. Methods of getting the material into solution are described.

John C. Long.

Minnes, James F. **The therapeutic evaluation of cortisone applied topically in ophthalmic diseases.** Canad. M.A.J. **66**: 342-347, April, 1952.

Topical cortisone is most effective in acute bacterial infection, allergy, burns, and traumatic conditions of the anterior segment. In burns of the anterior segment it limits corneal scars and symblepharon. The drug is indicated in extracapsular cataract surgery after the seventh postoperative day.

Irwin E. Gaynon.

Moser, R. **Dangers entailed in cortisone therapy.** Ophthalmologica **123**:313-316, April-March, 1952.

After prolonged local treatment with cortisone severe keratitis with hypopyon occurred as an acute complication in two cases of herpes simplex and in three cases of Fuchs's corneal dystrophy. The patients with herpes were under the effect of topical aureomycin therapy when the complication set in. Those with Fuchs's dystrophy had shown marked visual im-

provement up to the time of the acute keratitis. Bacteriologic studies were apparently not made. The cortisone was discontinued and intensive antibiotic therapy instituted whereupon the keratitis subsided very slowly. Cortisone is primarily indicated in the diseases characterized by low virulence of the bacterial invador and excessive inflammatory reaction of the invaded tissue. Cortisone is contraindicated in the diseases characterized by high virulence of the invader and weak response of the invaded tissue. The author warns against the use of cortisone in herpes simplex, that is, in dendritic keratitis. In disciform keratitis, however, cortisone works very well. Peter C. Kronfeld.

Mueller, P. **The indications for and the practical use of cortisone.** Ophthalmologica **123**:310-313, April-May, 1952.

In many forms of severe keratitis, iritis, or iridocyclitis, local cortisone administration combined with specific therapy directed at the etiology produces cures in a very much shorter time and with much less permanent tissue damage than any other known form of therapy. Interstitial keratitis is a striking example of such severe keratitis. Cortisone does not take the place of etiologic therapy, but demonstrates very convincingly the need for and the value of simultaneous etiologic therapy (removal of foci, antibiotics, sulfonamides). Adequate dosage of cortisone is very important. Many so-called cortisone-resistant patients get well on larger doses. Subconjunctival and systemic administration may become necessary in severe inflammations of the posterior segment or in panuveitis. Gradual withdrawal is important. In a case of penetrating injury, intraocular foreign body and very turbid media the topical administration of cortisone combined with the topical and systemic administration of antibiotics produced rapid clearing of the media which made possible visualization, localization and atraumatic extraction of the foreign body. In a case of severe chronic keratitis and iridocyclitis the topical administration of cortisone brought the inflammatory process to a standstill and held it at bay while daily injections of hypertonic sodium chloride were given retrobulbarly to promote clearing of the vitreous. During this treatment the vision improved from 3/60 to 0.4. The same form of treatment (cortisone topically) seemed to be beneficial in cases of cystic degeneration of the macula secondary to anterior uveitis. Peter C. Kronfeld.

Piekarski, Gerhard. **Serology of toxoplasmosis.** Klin. Monatsbl. f. Augenh. **119**: 1-11, 1951.

The author discusses the serologic tests (neutralization test on the rabbit skin, complement fixing test and the dye test of Sabin-Feldman) and their applications. Frederick C. Blodi.

Reese, A. B., Blodi, F. C., Locke, J. C., Silverman, W. A., and Day, R. L. **Results of use of corticotropin (ACTH) in treatment of retrolental fibroplasia.** A.M.A. Arch. Ophth. **47**:551-555, May, 1952.

In contrast to the promising results previously reported by the same authors from the use of corticotropin (ACTH) in the early progressive stages of retrolental fibroplasia, they now conclude that it has no effect on the course of the disease, and are discontinuing its use.

The cases discussed in this report were seen at Babies' Hospital and Lincoln Hospital in New York City. The criteria for instituting treatment were the occurrence of tortuosity of the retinal vessels, neovascularization, and early progressive edema in the periphery of the fundus. Many babies, premature or full-term, have a transitory gray zone in the far periphery of the fundus and this can easily be con-

fused with beginning retinal edema and detachment in the acute phase of retrolental fibroplasia. The average daily dose was about 15 mg. per kilogram of body weight per day and treatment was given for two to three weeks. After two weeks numerous complications were noted, notably diarrhea and pneumonias. Infants under treatment have a ravenous appetite, are irritable, and show inhibition of growth. R. W. Danielson.

Rintelen, F. **The prevention of hemorrhages during and after globe-opening operations.** Ophthalmologica **123**:228-235, April-May, 1952.

Some postoperative hemorrhages into the anterior chamber can only be explained as the result of an individual predisposition, which in some cases is revealed by the history (spontaneous hemorrhages from the nose or gums or previous postoperative hemorrhages). The recognition of this predisposition before cataract operation would be of great value. The results of bleeding time determinations do not show any relation to postoperative hemorrhages. Various forms of antihemorrhagic therapy (calcium, ascorbic acid, vitamin K, rutin) shorten the bleeding time but bring about no appreciable change in the incidence of postoperative hemorrhages. A fairly close relationship seems to exist between a predisposition to bleeding and a high sedimentation rate. Complete iridectomies are attended with a higher rate of hemorrhages than peripheral (not basal) iridectomies.

In the discussion, Thiel confirmed the lack of correlation between hematologic tests and bleeding tendency after globe-opening operations and stressed the occurrence of these hemorrhages during the fourth or fifth postoperative night. Other speakers mentioned the topical administration of thrombin as a means of preventing hemorrhage. Boehringer reported favorable results with the preoperative and postoperative systematic administration of a hemostatic epinephrine-derivative of the name of adrenoxyl. He had also observed an effect of sudden climatic changes upon the incidence of postoperative hemorrhages. Peter C. Kronfeld.

Ross, Milton G. **Some aids in ophthalmoscopy.** A.M.A. Arch. Ophth. **46**:637-646, Dec., 1951.

Aberration at the periphery of the cornea is an important factor in the blurring noted in the peripheral part of the fundus in direct ophthalmoscopy and may be eliminated through the use of the Troncoso contact glass. The ciliary body shadow can often be seen in great detail on the external surface of the globe when anterior scleral transillumination is used. Direct ophthalmoscopic transillumination is advantageous in examination of the posterior part of the fundus. A method of transillumination with indirect ophthalmoscopy for observation of the anterior part is described. A simple commercially available headlight may be used as an electric ophthalmoscope in the indirect method. (7 figures, 14 references)
John C. Long.

Saubermann, G. **The importance of the mode of administration in antibiotic therapy.** Ophthalmologica **123**:303-309, April-May, 1952.

Swiss preparations of penicillin have become more consistent with regard to potency and stability. For the treatment of serpentic ulcers the author prefers the iontophoretic administration of penicillin; in intraocular injections he resorts to subconjunctival injections. Sulfonamides and penicillin are compatible and may be true synergists in mixed infections.
Peter C. Kronfeld.

Strampelli, B. **A contact lens constructed to secure the penetrating corneal transplantation.** Boll. d'ocul. **31**:89-95, Feb., 1952.

In order to avoid opening the wound during the removal of sutures, the author advocates the use of a contact lens with a small window, which rotates along the sutured wound after penetrating keratoplasty. The lens is held by a ring-like retainer with four small holes which permit the fixation of the ring to the recti muscles. (6 figures) K. W. Ascher.

Strampelli, B. **A contact lens to be sutured to the eye muscles for the purpose of holding lamellar corneal transplants.** Boll. d'ocul. **31**:85-88, Feb., 1952.

Three drawings show 1. the contact lens, slightly smaller than the cornea, with four small holes near the lens border, 2. the fixation of this lens to the tendons of the four rectus muscles, and 3. an eye which had been operated upon with the lens in situ, fixed by four black silk sutures. K. W. Ascher.

Thorpe, H. E. **A double-mirror gonioprism.** Tr. Am. Acad. Ophth. **56**:97-99, Jan.-Feb., 1952.

The instrument consists of a truncated, quadrilateral, transparent, plastic pyramid which is mounted and easily rotated in an Allen plastic speculum. The pyramid has a concave corneal contact surface and there are two polished reflecting surfaces on two of its diametrically opposite inclined sides. The base of the pyramid is flat, and faces the observer during use. The anterior chamber angle is mirrored in the two reflecting surfaces of the prism. It can be studied with either the high or the low power of the slitlamp microscope. A stereoscopic tridimensional concept of the chamber angle structure and pathology can readily be obtained in this way. When the instrument is inserted between the lids with the truncated concave surface in contact with the cornea and the mirror surfaces facing in vertical axis, the simultaneous examination of the superior and inferior quadrants of the chamber angle becomes possible. By rotating this double reflecting contact prism through an arc of 90° about its anteroposterior axis, it also is possible to examine the nasal and temporal segments of the filtration angle.

The cornea is anesthetized with ½-percent pontocain. A drop of 1-percent methyl cellulose solution is placed in the corneal depression of the gonioprism. The patient is seated at the slitlamp microscope. His head is tilted backward 15° and he is instructed to rotate his eyes downward. The gonioprism with speculum handle pointing downward is then inserted between the lids in the same manner as a contact lens. It is slipped under the upper lid while the patient is looking downward. The lower lid is retracted and the speculum is inserted beneath the lower lid, and against the globe. As the last maneuver is completed the patient is instructed to gaze directly ahead. This brings his cornea in contact with the liquid film in the concavity of the gonioprism. After the observations with the slitlamp are completed, the instrument is removed in the reverse manner. After use, the methyl cellulose is wiped from the instrument with a damp, soft cotton cloth. The device may be sterilized by immersion in zephiran solution (1:2000) for five minutes, then rinsed and dried. (4 figures) Theodore M. Shapira.

Thorpe, H. E. **A new angled corneal curette.** Tr. Am. Acad. Ophth. **56**:99, Jan.-Feb., 1952.

A small corneal curette, 1 mm. in diameter, with an angled shaft, has been constructed. This instrument is useful for the removal of corneal foreign bodies or for curettage of embedded rust and of charred emery incrustations. The curette

is angled to the right for right-handed surgeons, and one is angled to the left for the left-handed individual. (1 figure)

Theodore M. Shapira.

Weidmann, W. **A new adaptometer.** Ophthalmologica **123**:355-356, April-May, 1952.

The test object, a ground glass illuminated from behind, is mounted on one end of a long horizontal cylinder which is kept in rotation around its long axis and covered with graph paper on its outside. The brightness of the target is varied by moving the light source within the cylinder. This is done by means of a handle which is coupled with a writing instrument on the outside of the cylinder. The exact position of the light source at any moment can thus be recorded on the graph paper covering the cylinder. The cylinder is long enough to permit reduction of target brightness down to $\frac{1}{100{,}000}$ of the initial value.

Peter C. Kronfeld.

Witmer, R. **The sero-test for tuberculosis of Middlebrook and Dubos.** Ophthalmologica **123**:329-334, April-May, 1952.

In his work on ocular tuberculosis the author has made extensive use of the hemagglutination test for tuberculosis devised by Middlebrook and Dubos (J. Exp. Med. 88:521, 1948). It is apparently a typical antigen-antibody reaction in which the antibody is a gamma-globulin. The results of the tuberculin test and of the hemagglutination test correlate well. It is advisable to do the hemagglutination test, that is the withdrawal of blood for the test, prior to the tuberculin test. There is a fair degree of parallelism between clinical signs of ocular or extraocular tuberculosis and the result of the agglutinin test. Patients with strongly positive hemagglutinin tests respond, on the whole, better to treatment with para-aminosalicylic acid than those with low agglutinin titer. The agglutinin test can also be done with the aqueous of diseased human eyes; there it proves the penetration of gamma-globulin into the aqueous in certain forms of inflammation.

Peter C. Kronfeld.

6

OCULAR MOTILITY

Burian, Hermann M. **Scope and limitations of orthoptics in nonsurgical treatment of ocular deviations.** A.M.A. Arch. Ophth. **45**:377-381, April, 1951.

Orthoptic treatment can do away with improper fixation, suppression and anomalous correspondence. It can develop proper visual habits and assist in the training or development of certain visuomotor functions. It may be a valuable adjunct to the surgical therapy of strabismus. Orthoptic training cannot "cure" a strabismus or compel someone to fuse or perceive stereopsis who does not have the faculty of sensory fusion or of stereopsis. Not every patient who may seem to be amenable to orthoptic treatment should be given exercises without further consideration. The ophthalmologic, social and educational indications and contraindications must be carefully weighed in each case.

John C. Long.

Franceschetti, A. **Acute concomitant strabismus.** Ophthalmologica **123**:219-226, April-May, 1952.

The author reports five cases of convergent strabismus characterized by the following features: 1. sudden onset of esotropia and diplopia, 2. a high degree of constancy of the angle of squint in the various positions of gaze, 3. no tangible signs of muscular palsy or of spasm of convergence, 4. excellent fusion, 5. no significant refractive error, 6. probably a preexisting exophoria and 7. excellent cosmetic and functional results achieved by small surgical interventions.

The mechanism of this form of eso-

tropia is not definitely established, but is probably not paralytic. The author favors the idea of a temporary impairment, by systemic disease, physical or psychologic trauma or unusual physical stress, of the fusion or divergence function followed by an acute escape reaction, namely esotropia. Prompt surgical treatment is advocated if conservative measures, including orthoptics, do not bring about a cure within one or two months.

Peter C. Kronfeld.

Fry, W. E., and Schlezinger, N. S. **Variable ophthalmoplegia due to angioneurotic edema.** A.M.A. Arch. Ophth. **46**:655-660, Dec., 1951.

A 25-year-old woman for several years experienced once or twice a month episodes of edema of the eyelids and unpredictable deviation of the involved eye in various directions. During the past year ocular deviation and diplopia has been present more or less constantly. The deviation of the affected eye often changed its direction during the night. This variable ophthalmoplegia is considered to be a manifestation of angioneurotic edema. The differential diagnosis and treatment are briefly considered. John C. Long.

Kretzschmar, S., Jr. **The measuring and plotting of ocular deviations by means of the perimeter of Goldmann.** Ophthalmologica **123**:348-351, April-May, 1952.

The addition of a second target projector makes Goldmann's perimeter well suited for the measuring and plotting of ocular deviations by the Hess method, known in the U. S. A. as Lancaster's red-green test (Arch. Ophth. *22*:867, 1939).

Peter C. Kronfeld.

Portmann, V. P., and Dufour, M. **The place of orthoptics in the treatment of convergent concomitant strabismus.** Ophthalmologica **123**:294-303, April-May, 1952.

This report from the eye clinic of the University of Lausanne is essentially a statistical study of the value of orthoptics in the treatment of convergent concomitant stabismus with no accommodative component or only a very slight one. The guiding principles were those developed and formulated by Chavasse. The results of surgery plus orthoptics were, on the whole, better than those of surgery alone. Attempts at normalization of retinal correspondence (by orthoptics) are not always indicated, especially not in adults whose strabismus started at a very early age and in whom one must suspect that the binocular reflexes developed only partially and are probably extinct. Orthoptics in these cases may give rise to very annoying and persistent diplopia. In the usual childhood form of esotropia the authors stress the value of the earliest possible recognition of the strabismus and the value of early inception of treatment.

Peter C. Kronfeld.

Rossetti, D., and Fregnen, E. **Ocular muscular equilibrium.** Rassegna ital. d'ottal. **21**:16-35, Jan.-Feb., 1952.

With the cylinder and the tangent scale of Maddox placed at five meters for examination at distance, and the Maddox wing test with relative prismatic correction for near, the authors studied the incidence of total and stato-neurogenic esophoria. This included the variations of esophoria in the transition of vision from distance to near. The authors value the Graefe test combined with Risley's rotary prism for determining the condition of the fusion impulse or the Maddox cylinder for estimating the functional condition of the extrinsic muscles. Eugene M. Blake.

7

CONJUNCTIVA, CORNEA, SCLERA

Chang C., and Chang H. L. **Epidemic kerato-conjunctivitis in Peking.** Chinese M. J. **69**:488-494, Nov.-Dec., 1951.

The authors report an epidemic of ker-

atoconjunctivitis in Peking during 1950 and 1951. The highest incidence was in the third decade. Seventy-two percent of the patients developed superficial punctate keratitis which developed 5 to 10 days after the onset of the conjunctivitis. Aureomycin, chloromycetin, penicillin, and sulfa drugs were of no use. Convalescent blood was effective.

Irwin E. Gaynon.

Duc, Camillo. **Rare orbito-conjunctival localization in lymphoid leukemia.** Rassegna ital. d'ottal. **21**:44-54, Jan.-Feb., 1952.

The leukemic process may express itself in various changes in the eyeball and its adnexa. It may involve only the fundus or the cornea and conjunctiva, or both. The author describes a 35-year-old man who had diffuse glandular enlargements and the blood picture of lymphoid leukemia when first seen. Later, lymphomatous masses appeared in the conjunctiva; there was a marked resemblance to vernal catarrh. An orbito-nasal mass was also observed. (4 figures, 15 references)

Eugene M. Blake.

Friede, Reinhard. **Surgery of the late stages of pemphigus of the conjunctiva.** Arch. f. Ophth. **151**:574-580, 1951.

Implants transferred from the lips or the skin to the conjunctiva for the relief of pemphigus were unsuccessful.

Ernst Schmerl.

Friede, R. **A new therapy for luetic interstitial keratitis.** Klin. Monatsbl. f. Augenh. **119**:23-41, 1951.

The author assumes that accumulation and stasis of toxins in the central part of the cornea are a main factor in the pathogenesis of interstitial keratitis. He, therefore, tries to dehydrate the corneal stroma by intracorneal and perilimbal injections of hypertonic glucose solutions, by desiccation of the corneal surface with heat and by paracenteses. In addition he uses penicillin intracorneally and advocates systemic treatment of the lues.

Frederick C. Blodi.

Leibiger, W. **A circumscribed bloodstaining of the cornea after a deep corneal hemorrhage.** Klin. Monatsbl. f. Augenh. **119**:76-79, 1951.

The author reports a case of a small circumscribed area of bloodstaining following a hemorrhage into the cornea from a deep vessel. Frederick C. Blodi.

Lijo Pavia, J. **Protected keratoplasty.** Rev. oto-neuro-oftal. **26**:123-129, Nov.-Dec., 1951.

The author reports seven cases treated with his new method of superficial keratectomy. A plastic contact lens protects the wound for 3 to 30 days.

Frederick C. Blodi.

Lijo Pavia, J. **Regeneration of the cornea without transplant.** Rev. oto-neuro-oftal. **25**:75-78, Aug.-Oct., 1950.

This is the preliminary report of an experimental study. With a 5-mm. trephine the center of the cornea of a rabbit was excised. Three preplaced episcleral sutures at the limbus were tied and the lids sutured. The defect was closed within 26 days by a transparent, epithelialized tissue. There was a temporary cloudiness and vascularization of the new cornea.

Frederick C. Blodi.

Messikommer, W. **Acute keratoconus in a child.** Ophthalmologica **123**:326-329, April-May, 1952.

The report concerns a seven-year-old boy in whom keratoconus, that is, the change from normal shape and thickness to marked conicity and apical thinning, occurred very rapidly in both eyes and was soon superseded by acute imbibition and clouding of the apex. The imbibition cleared slowly leaving behind dense corneal opacities. Peter C. Kronfeld.

Nelson, C. B., Pomeroy, B. S., Schrall, K., Park, W. E., and Lindeman, R. J. **An outbreak of conjunctivitis due to Newcastle disease virus (NDV) occurring in poultry workers.** Am. J. Public Health **42**: 672-678, June, 1952.

Newcastle disease of fowls was first recognized as a human infection when conjunctivitis caused by this agent was reported in 1943. Nine cases have been published where the virus was isolated. The authors report 40 cases among 90 workers in the eviscerating department of a chicken canning factory. Four of ten cases seen early yielded Newcastle disease virus. Most cases were unilateral, and preauricular lymphadenitis occurred in about one half of the cases. The symptoms were mild and lasted three to four days. No significant changes were found in acute or convalescent sera. However, the serum neutralization index was high in 64 percent of exposed workers who had no history of conjunctivitis.

Robert A. Moses.

Tóth, Zoltán. **Phylcentular keratoconjunctivitis and helminthiasis.** Klin. Monatsbl. f. Augenh. **119**:16-19, 1951.

In two girls phyctenular keratoconjunctivitis improved strikingly after intestinal parasites (oxyuris and tapeworm respectively) had been removed.

Frederick C. Blodi.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Amsler, M. **Cysts of the iris.** Ophthalmologica **123**:240-243, April-May, 1952.

The diagnosis of spantaneous cyst of the iris was made in three cases. It was based chiefly on the findings revealed by transillumination with the light source placed against the sclera on the side opposite the tumor. The area of the tumor was no darker than the rest of the iris nor did it connect with the ciliary body. In two of the cases the tumor definitely regressed during a period of several years.

Peter C. Kronfeld.

Amsler, M., and Huber, A. **Pertaining to the technique of and the principles underlying our fluorescein test.** Ophthalmologica **121**:130-136, Feb.-March, 1951.

The authors review and sum up the findings obtained with their fluorescein test which consists of the determinations by an optical method, of the rate of appearance in the anterior chamber of intravenously administered fluorescein (Am. J. Ophth. 30:1042, 1947, 31:1024, 1948). The test indicates the prevailing state of permeability of the blood-aqueous barrier, with sufficient accuracy for clinical purposes. In normal eyes, variations may be due more to extraocular than to intraocular factors. A fluorescein entry curve of a diseased eye is readily recognizable as such if it differs from the curve of its healthy mate. The fluorescein test is of great clinical value in the follow-up of cases of uveitis, contusion of the globe and perforating injuries.

Peter C. Kronfeld.

Note: This abstract was garbled when first printed in December, 1951.

Bruce, G. M. **Aberrant glandular tissue in the iris.** Tr. Am. Acad. Ophth. **56**:47-51, Jan.-Feb., 1952.

The author presents the case of a two-months-old boy, examined March 29, 1951. He was the fifth child and was born two weeks prematurely following an uneventful pregnancy. At the time of birth a small gray spot was noticed on the iris of the left eye. The mother thought it had become pinker and more elevated, and more oval in shape. When examined the tension in the left eye was normal; the cornea was the same size as the right. There was a pinkish-white tumor in the

iris from 7 to 9 o'clock at the periphery and extending toward the pupil as a rounded mass. Between the temporal side of the tumor and the pupil there was a 2-mm. strip of apparently normal iris tissue. The tumor, while regular in outline, had an irregular surface marked by numerous blood vessels collected in papilliform arrangement. A small area of the tumor, about 1.5 mm. in diameter near the lower pole, and another about 1 mm. in size near the upper pole, appeared to be cystic. The interior of the eye was normal. The tumor was easily removed by an ab externo limbal incision, and there have been no recurrences or sequelae. A pathologic study revealed a congenital anomaly consisting of aberrant glandular tissue in the iris. (7 photographs)

Theodore M. Shapira.

Cohen, Martin. **Iris cyst following traumatic implantation of glass splinter.** A.M.A. Arch. Ophth. **45**:413-415, April, 1951.

An injury resulted from a fragment of glass which had probably been retained in the eye for 45 years without causing deterioration of vision or changes in the fundus. There was a multiocular cyst in the lower sector of the anterior chamber close to the cornea. It is thought that implantation of the iris cyst is due primarily to the proliferation and migration of epithelial cells from the site of the perforation into the anterior chamber and iris. A secondary acute congestive glaucoma developed. This was controlled by miotics and was of short duration resulting in no visual damage. The noninflammatory reaction of glass in the eye is due to the fact that approximately 98 percent of glass is made up of neutral sodium silicate, which is chemically inert. John C. Long.

Cowper, Alexander R. **Harada's disease and Vogt-Koyanagi syndrome.** A.M.A. Arch. Ophth. **45**:367-376, April, 1951.

A 27-year-old Negro woman had symptoms of meningismus associated with bilateral uveitis, retinal detachments, scleritis with staphylomas, and extraocular muscle imbalance. There was some auditory disturbance and possibly a neurogenic bladder disorder. A positive electroencephalogram suggested diffuse cerebral involvement such as is found in meningoencephalitis. Many aspects of the case placed it somewhat between Harada's disease and the Vogt-Koyanagi syndrome. The author describes these conditions in some detail, including symptomatology and possible etiology. He feels that they are both manifestations of the same disease and should be regarded as such. The name uveoencephalitis is proposed for this disease entity.

John C. Long.

Hoffman-Egg, Lilly. **Sympathetic ophthalmia after iridencleisis.** Ophthalmologica **123**:207-211, April-May, 1952.

While recovering from pneumonia, the 72-year-old patient developed a bilateral acute glaucoma which proved refractory to all conceivable forms of treatment including one iridencleisis, one typical cyclodiathermy after Vogt and one retrociliary diathermy after Weekers on each eye. The pneumonia recurred and both eyes became blind and remained painful; first the right, and, three months later, the left eye was enucleated. The right eye showed a very striking degree of retinal perivasculitis with nodular thickenings which consisted of plasma cells, lymphocytes and epithelioid cells. The lens was incarcerated in the iridencleisis incision. In the left eye, the anatomical picture was that of a panophthalmitis which had become manifest clinically during the last few days preceding the enucleation. The author interprets the disease of the right eye as sympathetic ophthalmia.

Peter C. Kronfeld.

Kellerman, Leo. **Pregnenolone therapy in uveitis.** A.M.A. Arch. Ophth. **46**:419-420, Oct., 1951.

Pregnenolone is chemically related to the adrenal corticosteroids. This drug was given to ten patients with various types of active uveitis. There were no toxic reactions but nine of the ten patients failed to show any change.

John C. Long.

Lijo Pavia, J., Arouh, J., Lis, M. **A rare congenital anomaly of the choroid.** Rev. oto-neuro-oftal. **25**:62-64, June-July, 1950.

A round lesion, the size of 3 DD was found accidentally in the nasal periphery in the fundus of one eye of a 55-year-old woman. The lesion had a yellow-white color and was covered with normal retina. It consisted of contracted vessels arranged like an arbor vitae. A similar case, described by E. Fuchs, appears in Dimmer's atlas and was thought to be an atypical coloboma. The authors believe it is the sequel of an angioma.

Frederick C. Blodi.

Streiff, E. B., and Rosselet, E. **The relationship between penetrating injuries, foci of infection and sympathetic ophthalmia.** Ophthalmologica **123**:235-240, April-May, 1952.

The clinical course after perforating injuries of the eye is extremely variable. The early subsidence of all inflammatory symptoms followed by flare-ups several months or years later is especially difficult to understand. The authors reviewed all cases of perforating injury treated at the University Eye Clinic of Lausanne as to their clinical course, the presence of extraocular foci of infection, and the results of the usual laboratory tests. Special attention was paid to the presence of tuberculosis. This review, while not in any way conclusive, suggested that the patient's general state of health and particularly the presence of foci of infection played a role in the evolution of the posttraumatic inflammatory processes. To the prevailing theories of the pathogenesis of sympathetic ophthalmia, the authors add the concept of the traumatized uvea representing a locus minoris resistentiae and thereby becoming susceptible to bacterial allergens liberated as the result of a disease which had existed before or was acquired after the ocular injury. Such a concept would account for the wide variations in the severity or course of posttraumatic inflammations. The second eye may become involved in the process through sensitization of its uvea as the result of allergens liberated by the uvea of the injured eye. The involvement of the second eye may also come about through the neuro-vascular relationship between the two eyes demonstrated by the fluorescein test (Am. J. Ophth. 33:326, 1950).

In the discussion Verrey stated that the extremely variable clinical course of sympathetic ophthalmia was a strong argument against a single etiology. Examination of the aqueous of two sympathizing eyes revealed phagocytes laden with bacteria.

Peter C. Kronfeld.

Sun, K. Y. **Congenital aniridia.** Chinese M. J. **69**:495-496. Nov.-Dec., 1951.

The author reports a case of bilateral congenital aniridia in a 20-year-old Chinese girl and discusses the etiology and developmental pathology. Tatooing at the periphery of the cornea, or use of a contact glass is advocated for treatment.

Irwin E. Gaynon.

Winter, F. C., and Maumenee, A. E. **An unusual pigmented tumor of the iris.** A.M.A. Arch. Ophth. **46**:438-440, Oct., 1951.

A girl, aged 7 years, had had a small black spot on the iris since the age of three months. The lesion had become progressively larger and finally extended from the limbus to the pupillary margin,

touching the cornea. An excision for biopsy was done and later an enucleation. The tumor was thought to have arisen from the branched chromatophores of the iris. The child is now in good health.

John C. Long.

Witmer, R. **A special form of recurrent choroiditis.** Ophthalmologica **123**:353-354, April-May, 1952.

The author describes a recurrent, relatively benign choroiditis, with recurrences which are all located fairly close to the original lesion or, rather, to a precipitate-like gray mass on the posterior retinal surface to which the choroidal recurrences seem to be closely related. The systemic findings and laboratory tests spoke for tuberculosis. The recurrences seemed to be favorably influenced by paraaminosalicylic acid by mouth and streptomycin subconjunctivally. Peter C. Kronfeld.

Witmer, R. **Chronic uveitis associated with bronchiectasis.** Ophthalmologica **123**:352-353, April-May, 1952.

In four cases of chronic uveitis the systemic findings were as follows: tuberculin and seroreactions for tuberculosis (Middlebrook and Dubos) negative or very weakly positive; mild eosinophilia of the blood; chest findings suggestive of chronic bronchitis; bronchiectasis revealed by bronchography. All four patients were very favorably influenced by penicillin-streptomycin inhalations.

Peter C. Kronfeld.

Witmer, R. **Electrophoretic studies of human aqueous.** Ophthalmologica **123**: 280-284, April-May, 1952.

Electrophoresis, that is, the determination of the rate of migration of protein particles under the influence of a strong direct current, is a method for the identification of protein entities within a protein mixture (von Sallmann, Arch. Ophth. 40:279, 1948). Applied to the aqueous of human eyes with acute or chronic uveitis the method revealed essentially the same distribution of the various protein fractions as in standard human blood plasma. As Goldmann stated in the discussion, the permeability of the blood-aqueous barrier of the inflamed eye is probably increased to the same extent for both albumins and globulins. Locally produced or liberated cell proteins contribute only slightly to the proteins of the aqueous.

Peter C. Kronfeld.

9

GLAUCOMA AND OCULAR TENSION

Bedrossian, E. H. **Increased intraocular pressure secondary to mediastinal syndrome.** A.M.A. Arch. Ophth. **47**:641-642, May, 1952.

A 66-year-old man had swelling of the entire head, face, and neck and bilateral glaucoma, secondary to interference with circulation from a mediastinal tumor. The glaucoma was secondary to obstruction of both the cavernous sinus and the jugular drainage systems, but the intraocular pressure could, nevertheless, be reduced to normal by pilocarpine.

R. W. Danielson.

Boehringer, H. R. **Secondary glaucoma with neovascularization on the iris.** Ophthalmologica **123**:211-215, April-May, 1952.

All diseases of the posterior segment which involve retinal vessels to a considerable degree (diabetic or hypertensive retinopathy, obstruction of the central retinal vein, infectious periphlebitis, or Coats's disease) may lead to secondary glaucoma with neovascularization in the iris for which there is no effective therapy.

Peter C. Kronfeld.

Brand, Imre. **The influence of placental extracts on the intraocular pressure.** Klin. Monatsbl. f. Augenh. **119**:47-55, 1951.

Placental extracts have little influence on eyes with a normal pressure. They may cause an increase in tension in primary glaucoma. In cases of secondary glaucoma placental extracts seem to lower the increased pressure.

Frederick C. Blodi.

François, J. **Gonioscopy and vascular hypertension.** Rassegna ital. d'ottal. **20**: 257-258, Sept.-Oct., 1951.

François reminds us that the study of the corneo-irideal angle does not solve the problem of glaucoma. Such changes as pigmentation of the trabeculum and Schlemm's canal do not differ from those which may be seen in normal eyes 50 years of age. Congenital glaucoma is characterized by the presence of embryonal mesodermic tissue. This differentiates it from the juvenile type and from true megalocornea. The latter does exist but most cases which are reported as such are really incomplete congenital glaucoma. Congenital mesodermal tissue is usually found in aniridia, coloboma of the iris and in microphthalmos. The writer then discusses the well-known changes in hypertensive uveitis, traumatic, post-operative and hemorrhagic glaucoma and rubeosis iridis.

Eugene M. Blake.

Gasparri, F. **Prothrombinemia in primary glaucoma (study of 100 patients).** Boll. d'ocul. **31**:175-185, March, 1952.

One hundred patients suffering from primary glaucoma (three tables) were subjected to prothrombin studies. Seventy-one showed an increase of their prothrombin rate, fifteen a decrease, and fourteen had normal values. Endothelial changes in the smaller blood vessels, particularly of the brain, may be responsible. (References)

K. W. Ascher.

Goldmann, H. **The mode of action of the iridectomy in chronic congestive glaucoma.** Ophthalmologica **123**:202-206, April-May, 1952.

By means of a method based principally on measurements of the rate of entry of intravenously injected fluorescein (Am. J. Ophth. 33:1630, 1950) Goldmann has been able to estimate the rate of aqueous flow through the anterior chamber (or minute volume) of the human eye. Simultaneous measurements of the ocular tension and of the pressure in the anterior ciliary veins yield the so-called outflow pressure, that is, the amount of hydrostatic pressure necessary to overcome the resistance between anterior chamber and extraocular veins. Dividing this outflow pressure by the minute volume gives an expression of the amount of resistance. The results for three groups of human eyes are shown in the following:

	outflow pressure mm. Hg.	minute volume cu. mm.	resistance
Normal	6.5	2.2	3.0
Simple glaucoma	28	1.67	18
Chronic congestive glaucoma	38.5	4.1	10

The resistance to flow was found to be increased in both types of glaucoma, but more so in the noncongestive variety. The minute volume was lower than normal in simple glaucoma and definitely higher than normal in chronic congestive glaucoma. Gonioscopically, the latter cases were characterized by apparent extensive closure of the angle by the root of the iris. Since the minute volume was found increased in a large majority of these cases, Goldmann concludes that this apparent closure was not watertight. The increase in minute volume was probably related to the vascular reactions characteristic of congestive glaucoma.

After iridectomies consisting of complete excision of an iris sector of about

90 degrees (Parker, Arch. Ophth. 37:277, 1947) the minute volume dropped to normal. The resistance was also reduced, but usually remained above the normal range. The author concludes that the iridectomy acts primarily by reducing the minute volume. Peter C. Kronfeld.

Kleinert, Heinz W. **The compensation maximum.** A.M.A. Arch. Ophth. **46**:618-624, Dec., 1951.

In eyes with glaucoma a retrograde influx of blood from episcleral veins into aqueous veins occurs during compression of the eyeball. The dynamometer pressure necessary to produce this phenomenon is termed "compensation maximum." In eyes with normal pressure, compression up to even 150 gm. usually will not produce the backflow phenomenon into aqueous veins, while the compensation maximum of glaucomatous eyes in many instances is found to be below 100 gm. The appearance of the backflow phenomenon into aqueous veins depends upon an interference with aqueous-humor elimination. The readings are helpful in the diagnosis of suspected glaucoma.

John C. Long.

Rama, G. **Retrociliary diathermy and cyclodialysis in some forms of glaucoma.** Rassegna ital. d'ottal. **21**:36-43, Jan.-Feb., 1952.

Rama performed nonperforating cyclodiathermy combined with cyclodialysis in five cases of congenital glaucoma and in one of postcataract congestive glaucoma. The diathermy was performed in one hemisphere of the eye and repeated in the other in about two weeks. The dialysis was extensive and was made through two openings, one above and one below. Satisfactory and lasting reduction of tension was secured. Eugene M. Blake.

Robertson, E. Norris. **Surgical treatment of congenital glaucoma.** A.M.A. Arch. Ophth. **47**:611-615, May, 1952.

The author discusses the history and manner of surgery in goniotomy (Barkan) and goniopuncture as advocated by Scheie. One or both operations have been tried on eleven eyes with good results in six. R. W. Danielson.

Thiel, R. **The pathogenesis of primary glaucoma.** Ophthalmologica **123**:195-197, April-May, 1952.

The author assumes regulation of the intraocular pressure by a diencephalic center which is in rapport with the other vegetative centers and functions principally by mediating conpensatory mechanisms under conditions of stress. As evidence in favor of such a center, Thiel lists the tension-lowering effect of central depressants such as gynergen, dolantin and evipan, the tension-raising effect of central stimulants such as pervitin and isophen and the tension-lowering effect of ganglionic blocking agents such as pendiomide.

Primary glaucoma is probably related to the senile changes within the eye, which consist of overgrowth of connective tissue in the ciliary body, sclerosis of the corneoscleral trabeculum and aging of the vitreous. The increase of connective tissue in the ciliary body lowers the sensitivity of the peripheral sensory receptors of the regulatory mechanism.

Peter C. Kronfeld.

Zollinger, R. **Clinical investigations of neovascularization on the iris.** Ophthalmologica **123**:216-219, April-May, 1952.

Corroborating a conclusion reached previously on the basis of histologic examinations (Am. J. Ophth. 34:1784, 1951) the author finds that clinically the combination of secondary glaucoma with neovascularization on the iris is characteristic of cases with obstruction of the central retinal vein or of total retinal detachment.

Peter C. Kronfeld.

10

CRYSTALLINE LENS

Bellows, John G. **Lens and virteous.** A.M.A. Arch. Ophth. **45**:472-498, April, 1951.

The publications which appeared in 1949 and 1950 are reviewed. (146 references) John C. Long.

Diabetes and cataract. Resume of a conference in the Prof. J. Britto Ophthalmological Seminar in February, 1950. Arq. brasil. de oftal. **13**:179-180, 1950.

This is a summary of a conference during which the relationship between cataract and diabetes was discussed. It is believed that diabetes is a complex endocrinopathy and that the function of other glands of internal secretion affect glycemic equilibrium to some degree. A distinction between pituitary diabetes, which is insulin-resistant but sensitive to radiation of the hypophysis, and insulin sensitive pancreatic diabetes is made.

Two types of cataract occur in the diabetic. The usual "senile cataract" is a well known finding. On the other hand, the very rare true diabetic cataract may develop in a short time and completely mature within a few days. It is bilateral, may occur in young patients, and is associated with hypercholesterolemia. Treatment is prophylactic rather than curative, and includes diet, insulin, and hygienic measures. When the cataract has progressed sufficiently, surgical intervention is indicated. James W. Brennan.

Hruby, K. **Recommendations for cataract extraction.** Klin. Monatsbl. f. Augenh. **119**:121-135, 1951.

The author describes the technique used for the intracapsular extraction at Lindner's Clinic. The result of 646 extractions are given. Vitreous complications occurred in only 8 percent of the cases by using traction sutures in all rectus muscles. Expression is advised for intumescent cataracts. Frederick C. Blodi.

Malatesta, C. **Congenital cataract and other ocular and general malformations after German measles during pregnancy.** Boll. d'ocul. **31**:79-84, Feb., 1952.

A three-and-one-half-year-old girl, whose mother had had rubella during the first month of pregnancy had mental retardation, splenomegaly, spastic paresis of the left inferior extremity and congenital valvular disease as well as unilateral cataract. She was a deaf-mute. The eye with a transparent lens showed an atypical pigmentary degeneration. (49 references) K. W. Ascher.

Pau, Hans. **Nuclear cataract as a sign of degeneration of the globe.** Klin. Monatsbl. f. Augenh. **119**:12-16, 1951.

The author presents ten cases of various unilateral degenerative ocular diseases (myopia, diabetic retinopathy, glaucoma). In these eyes a brown nuclear cataract was present, probably caused by poor nutrition of the lens.

Frederick C. Blodi.

Siedenbiedel, H. **Circulation of fluid in the human lens.** Klin. Monatsbl. f. Augenh. **119**:79-81, 1951.

The liquid cortex of a hypermature lens showed continuous movement similar to caloric movement of the aqueous. This is the first observation of such a movement in an intact human eye. It could be well observed because of many suspended glittering crystals in the cortex.

Frederick C. Blodi.

Tyner, G. S., and Frayer, W. C. **Clinical and autopsy findings in early retrolental fibroplasia.** A.M.A. Arch. Ophth. **46**:647-654, Dec., 1951.

This is a clinical and pathological report on both eyes of a premature infant with bilateral retrolental fibroplasia in its early

stages. A total of 263 mg. of corticotropin was administered. During the treatment a generalized pustular eruption of the skin, some rounding of the facial contours, and prominence of the globes developed. Death was due to acute peritonitis. It was thought that the areas of retinal involvement did not increase after treatment with corticotropin. The principal pathologic changes were a heavily vascularized fibrous membrane, arising from the inner layers of the retina extending toward the center of the globe, small vascular buds from the vessels in the nerve fiber layer in the viterous, retinal folds, edema and proliferation of pigment epithelium of the retina, and excessive small, blood-filled vascular channels in the iris and ciliary body. (8 photomicrographs) John C. Long.

11

RETINA AND VITREOUS

Adelung, J. C. **The genesis of angioid streaks (Knapp).** Klin. Monatsbl. f. Augenh. **119**:241-250, 1951.

In the case reported, angioid streaks were combined with pseudoxanthoma elasticum of the skin (Groenblad-Strandberg-Syndrome). Granular degeneration of the elastic substance of the cutis has been reported in pseudoxanthoma and angioid streaks are produced by similar changes. The author uses a rubber ball model to demonstrate the role which mechanical factors play in the production of this condition. Accommodation is said to exercise a corresponding strain upon the elastic lamina of the choroid. Therapeutically, paralysis of the accommodation might be worth trying. Ernst Schmerl.

Amsler, M., and Hoffmann-Egg, L. **The anatomical findings in a case of layer-tear of the retina.** Ophthalmologica **123**: 269-271, April-May, 1952.

In the left eye of a 76-year-old man, a shallow, much folded and very opaque retinal detachment could be seen to occupy the inferotemporal half of the eyeground, with a dark-red sharply outlined lesion, resembling a tear, in the equatorial region. Opacities of the media precluded a more accurate ophthalmoscopic diagnosis. The patient died of a cerebral vascular accident before surgery could be undertaken. The eye became available for anatomical study. Macroscopic examination through a window made by removing a nasal calotte as well as microscopic examination revealed a so-called layer-tear in the retina, that is, extensive cystic distruction of the outer layers and only a tiny hole in the innermost, vessel-bearing layer. Peter C. Kronfeld.

Bailliart, P. **The retina and generalized arterial hypertension.** Rev. oto-neuro-oftal. **25**:125-128, Dec., 1950.

The report of an ophthalmologist on a hypertensive fundus when given to a general practitioner should be intelligible to the latter. Special attention should be paid to the tension in the central artery. If the tension is higher than normal (half of the diastolic pressure in the brachial artery), an increased pressure in the cerebral vessels must be assumed.

Frederick C. Blodi.

Dellaporta, A. **Shortening of the globe by episcleral resection.** Klin. Monatsbl. f. Augenh. **119**:135-140, 1951.

In 1950 the author first reported his experiences and carefully planned experiments on episcleral resections for certain cases of retinal detachment. The inner third of the sclera is not excised and becomes folded inward when the superficial layers are sutured. He gives here a detailed description of his technique, which has lately become popular in this country and in France. Frederick C. Blodi.

De Vries, S. **Retinopathy in dermatomyositis.** A.M.A. Arch. Ophth. **46**:432-435, Oct., 1951.

Dermatomyositis is a rare disease of unknown etiology, belonging to the group of collagenous diseases. A case of this disease is reported in a woman, 49 years of age. Both eyes showed a number of grayish-white patches and groups of small white spots in the nerve fiber layer of the retina. There were no retinal hemorrhages or changes in the retinal blood vessels. Death occurred from the systemic disease. Microscopic examination of the retina showed cytoid bodies in the nerve-fiber layer (varicose or ganglion-like swelling of fibers). The involved area of retina also showed an albuminous deposit and a separation of parallel nerve fibers.

John C. Long.

Duff, I. F., Falls, H. F., and Linman, J. W. **Anticoagulant therapy in occlusive vascular disease of the retina.** A.M.A. Arch. Ophth. **46**:601-617, Dec., 1951.

The prognosis for adequate visual acuity after untreated thrombosis of a tributary or central retinal vein is discouraging. In a control series of 79 untreated patients with complete occlusion of a central retinal vein, the condition of 49 grew worse, and secondary glaucoma developed in 43 percent. Only 15 percent of these patients showed improvement, whereas the condition of 35 percent remained unchanged. The prognosis appears to be much less grave for untreated incomplete thrombosis of a central retinal vein.

Experience with anticoagulant therapy after thrombosis of a tributary or central retinal vein indicates that in 58 percent the prognosis for visual improvement is favorably influenced; the frequency, moreover, of secondary glaucoma appears to have been encouragingly lowered. Under treatment, however, not all will recover. The visual acuity of 19 percent will remain unchanged, and that of 9 percent will definitely grow worse. A short term of intensive treatment with heparin appears to produce results as favorable as those obtained with prolonged use of bishydroxycoumarin (dicumarol). The incidence of hemorrhage in long-term treatment with bishydroxycoumarin, moreover is appreciable. Heparin is recommended for general use. (10 tables, 17 references)

John C. Long.

Goldmann, H., and Tobler, W. **The etiology of retrolental fibroplasia.** Schweiz. Med. Wchnschr. **82**:381-385, April, 1952.

The literature on the etiology and pathogensis of retrolental fibroplasia is reviewed and the first case of retrolental fibroplasia which occurred in the Infants Home in Berne (in 1951) is reported. Five hundred seventy premature babies with birth weights under four pounds had developed with normal eyes up to this time. The institution had acquired a new incubator a few months previously which made a constant oxygen concentration of 60 percent available to the infant. The second premature infant exposed to this high oxygen atmosphere also acquired the disease. The oxygen concentration has since been lowered to 30 percent and no new cases have been observed. The authors conclude that a high concentration may either be directly harmful, or the premature infant may adapt itself to the high concentration, but develop retrolental fibroplasia as a result of a quick changeover from the oxygen-rich atmosphere to one containing normal air. (References)

Max Hirschfelder.

Johnston, K. B. **The retinal vessels of the fundus oculi in hypertension.** Canad. M.A.J. **66**:233-234, March, 1952.

In teaching and in reports on consulta-

tions the staff members at McGill University use the terminology and classification as given by Nicholls in Stallard's textbook. The term retinopathy has replaced retinitis and is applied to a fundus in which hemorrhages, exudation or edema of the nerve head are seen. When there are vacular alterations only, the fundi are described as eyegrounds with retinal vascular changes (type and grade) without retinopathy.

Retinal vessels beyond the branching of the central artery and vein are termed arterioles and venules. Critical, detailed observation is made of the following characteristics: general arteriolar narrowing, focal constriction, and compression changes at the arteriolar-venule crossings. The changes found are classified as mild, moderate, marked, or severe. Loss of translucency, irregularity of light streak and copper wire or silver wire appearance suggest arteriolar sclerosis; it may be present with hypertensive vascular signs. When arteriolar sclerosis is present, the changes are permanent. Hypertensive changes alone may be spastic and in that case may regress. Grading ratios and brief descriptions of Gunn's and Salus' signs are given. F. M. Crage.

Lijo Pavia, J. **Complication after operations for retinal detachment.** Rev. oto-neuro-oftal. **25**:110-115, Nov., 1950.

The author discusses hemorrhages and thickening of the detached posterior hyaloid; both have a good prognosis. Two other complications, exudative and proliferative retinitis, were also encountered. Frederick C. Blodi.

Lijo Pavia, J. **Ophthalmoscopic and photographic aspects of the macula.** Rev. oto-neuro-oftal. **25**:29-35, March-May, 51-58, June, July, 85-91, Aug.-Oct., 1950.

The author described the ophthalmoscopic appearance of the macula and of the perimacular reflex caused by the unevenness of the retina and produced by the internal limiting membrane. Variations of the perimacular reflex are discussed. He next describes the macular and the foveal reflex, with the blood supply of this area and gives a description of the hypertonic macula. The advantages of ophthalmoscopy with red-free and monochromatic light are brought forward. There are fundus photographs and 22 references. Frederick C. Blodi.

Lijo Pavia, J. **A new simple classification of hypertensive retinopathies.** Rev. oto-neuro-oftal. **25**:6-13, Jan.-Feb., 1950.

The author proposes the following classification. 1. First grade. Vision is normal; there may be nasal pallor of the disc and sometimes a slight edema at the upper and lower margin; the retina may be edematous, especially in its macular area; the caliber of the retinal arterioles is decreased because of an increased tonus and the light reflex of the arterioles is increased (copper wire arteries). 2. Second grade. Central vision is normal, but the visual field shows atypical changes, usually a concentric depression; there is pallor of the disc with a grayish discoloration of the retina around it. Glittering white dots appear in the internal limiting membrane, cotton wool patches and small hemorrhages in the nerve-fiber layer; there is a marked attenuation of the arterioles to half of the caliber of the corresponding veins; the caliber of the arterioles may vary (angiospasm) and the light reflex is increased (silver wire arteries); the arterio-venous crossings show nicking of the veins and there is a sclerosis of the choroidal vessels often visible because of atrophy of the pigment epithelium; the blood pressure in the arterioles is lower than normal. 3. Third grade. Central vision is diminished, the blind spot enlarged and the visual field concentrically

depressed; there is papilledema with extensive retinal edema; the retina shows cotton wool patches, edema residues, glistening crystals and hemorrhages; the arterioles are maximally contracted with numerous foci of angiospasm; at the arterio-venous crossings the central part of the vein is dilated and hyperemic; the blood pressure in the arterioles is higher than normal and approaches the pressure in the brachial artery.

In the first grade, which usually occurs in patients below 50, the prognosis is good. The prognosis is guarded in patients with a retinopathy of the second grade; in the third grade, the disease is a malignant hypertension. Frederick C. Blodi.

Lijo Pavia, J., and Arouh, J. **Vitreous detachment in a case of luetic chorioretinitis.** Rev. oto-neuro-oftal. **26**:3-9, Jan.-Feb., 1951.

In discussing a case of posterior detachment of the vitreous the authors emphasize the importance of a careful examination of the vitreous. The various means of examining the vitreous are discussed. Biomicroscopy of the posterior segment of the eye is not mentioned.

Frederick C. Blodi.

Mancall, Irwin T. **Occlusion of the central retinal vein** (Ophthalmological Review) A.M.A. Arch. Ophth. **46**:668-676, Dec., 1951.

In occlusion of the central retinal vein it has been found that the obstruction is due to a true thrombosis, and that the thrombi may become canalized. It has also been demonstrated that occlusion may be the result of endothelial and subendothelial proliferation, with closure due to contraction of the new-formed connective tissue and endothelial proliferation into the lumen of the vein. There may be degeneration of the intima, with undermining of the degenerated intima by the blood stream of small collaterals of the vein, thus forming new channels in a manner similar to that of a dissecting aneurysm. In elderly arteriosclerotic patients the occlusion may be ascribed to endothelial and subendothelial proliferation. Cases associated with infections or septic emboli or hematologic diseases or trauma are probably true thromboses.

John C. Long.

Mattos, Rubens Belfort **Reflection about retinitis pigmentosa.** Arq. brasil. de oftal. **13**:145-150, 1950.

Retinitis pigmentosa may be considered a primary degeneration of the neuroepithelium, an abiotrophy, with a known hereditary factor. Tissue therapy with its assumed production of biogenic stimulators has been used by the author in five patients with this disease. The techique of Filatov was used. Such therapy is nonspecific, and the mode of action is unknown, although an enzyeme action, hormone, inhibition of carbohydrate synthesis, and lowering of surface tension have all been postulated. The tissue itself or an aqueous extract may be administered intramuscularly, subconjunctivally or retrobulbarly. Five patients were treated and observed over a period of several months; some degree of improvement in central acuity and in the peripheral field was noted in three. The author's preliminary impression was favorable. His method of preparing the tissue and aqueous extract is given briefly.

James W. Brennan.

Maumenee, A. E. **Diseases of the retina.** A.M.A. Arch. Ophth. **47**:643-690, May, 1952.

This 47 page, unusually exhaustive review of the literature of 1951 on the retina has been excellently done. All subjects are

covered, but the discussion of retrolental fibroplasia and of vascular diseases is emphasized. R. W. Danielson.

McGraw, James L. **Malignant melanoma associated with retinal hole.** A.M.A. Arch. Ophth. **46**:666-667, Dec., 1951.

The author presents an observation that demonstrates that a hole in the retina with a detachment and malignant melanoma can coexist. Originally it was believed that the patient had uncomplicated detachment of the retina with a hole. After diathermy the hole was sealed and the retina became reattached. A second hole developed, and the retina again became detached. Ultimately malignant melanoma of the choroid was found.

John C. Long.

Rundles, W. Z., Jr., and Falls H. F. **Congenital arteriovenous (racemose) aneurysm of the retina.** A.M.A. Arch. Ophth. **46**:408-418, Oct., 1951.

Congenital arteriovenus aneurysms of the retina may exist as an isolated entity or be associated with arteriovenous aneurysm or other vascular malformations in other locations, especially the brain. The 31 cases of this anomaly found in the literature between 1874 and 1951 are summarized in tabular form. Three new cases are reported. In two the anomaly was apparently confined to the retinal vessels; in the other, extensive intracranial vascular anomalies ultimately lead to death. The cerebral and ocular malformations and their embryologic basis are described. The ophthalmoscopic picture is not unmistakable. Vascular intercommunication may result from prolonged stasis. In any disease associated with greatly enlarged and tortuous vessels, arteriovenous communication must be excluded. The Sturge-Weber syndrome and angiomatosis retinae of v. Hippel may suggest arteriovenous aneurysm. John C. Long.

Schepens, C. L. **Subclinical retinal detachments.** A.M.A. Arch. Ophth. **47**:593-606, May, 1952.

A retinal detachment may be called subclinical when the diagnosis can be made only by unusual methods of investigation. The unusual methods described consist of Schepens' stereoscopic indirect ophthalmoscope and scleral pressure, prolonged study, very strong light, and study in a dark room with the patient sitting up and lying down. The visual field and the central acuity in such cases are not affected by the detachment unless other ocular abnormalities are present. The signs of a very flat and peripheral detachment are 1. ill-defined choroidal pattern, 2. a visible shadow of retinal vessels on the chorold, and 3. abnormal watered-silk-like reflections. Indirect illumination may give the flatly detached retina a characteristic cobblestone appearance. Special contact lenses have also been used to facilitate examination of the extreme periphery.

During a period of three years, 102 patients of the Retina Service of the Massachusetts Eye and Ear Infirmary were found to have a subclinical detachment with retinal break; 43 patients complained of blurred vision, floating specks, and flashes of light. Retinal break is a comprehensive term which includes tears (with operculum), holes (without operculum) and dialyses (disinsertions). Tears are more common at the equator, holes at the ora, and holes are about twice as common as tears. Forty percent of the eyes showed opacities of the posterior lens cortex. Thirty-nine eyes, or 35 percent, were operated upon and 38 had complete reattachment after operation; in 32 eyes surface diathermy alone was used, because of the close approximation, and only weak applications were made. (2 color plates, 7 tables).

R. W. Danielson.

Stallard, H. B. **Irradiation of retinoblastoma. (Glioma retinae)** Lancet 1: 1046-1049, May 24, 1952.

Retinoblastoma is uniquely a radiosensitive tumor arrising from radioresistant tissue. In 40 percent of cases the tumor is bilateral. Successful irradiation treatment is often possible when the tumor affects one third or less of the retina and has not invaded the optic disc. In more advanced cases the prognosis for vision is poor. Therefore, emphasis is placed on early diagnosis and adequate ophthalmoscopic follow-up.

Sections of successfully irradiated retinoblastomas show reduction in vascularity of choroid and retina. Changes in the vessels include varicosities, collapse, and degeneration of the adventitia and media. A few clumps of distorted, inactive retinoblastoma cells compressed by fibrous tissue were found. Such cells may remain inactive for years. Iridocyclitis and secondary glaucoma were less frequent after use of radon seeds and radium discs than after deep X-ray therapy.

Comparing his own series with those of A. B. Reese and others the author concludes that the best results are to be obtained with the use of radium discs shaped to fit the globe and fixed in place. Eighty-three percent of the patients so treated have retained some useful vision.

Attention is drawn to the remarkable development of ability to use the nasal portion of the retina for reading when the papillomacular region has been destroyed. This was observed in four patients. Robert A. Moses.

Streiff, E. B. **The present state of therapy in retinal detachment.** Rassegna ital. d'ottal. **20**:255-256, Sept.-Oct., 1951.

Streiff reviews the history of our knowledge of retinal detachment and confirms Gonin's belief that closure of the retinal tear is essential to recovery. He recommends control of the diathermy by use of the indirect method of ophthalmoscopy. At the present time 50 percent of cases of retinal separation are cured by proper diathermy. Other methods of treatment, such as chemical cautery, scleral resection and scleral folding are briefly considered. Eugene M. Blake.

Tsopelas, Basilios. **Hemorrhages into the macula from war-injuries.** Klin. Monatsbl. f. Augenh. **119**:298-307, 1951.

The newer weapons of war cause frequent direct, or more often, indirect hemorrhage into the macula.

Twenty-three cases of retinal hemorrhages were found in 297 persons with injuries of the head near the eyes. There was likewise hemorrhage into the vitreous in 11 cases, rupture of the choroid in 2, rupture of the macula in 2, luxation of the lens in 2 and retinal detachment in 1. The patients were followed for about three months. Sometimes chorioretinitis pigmentosa or chorioretinitis proliferans developed. Complete recovery occurred in 2 cases, 3 men retained a vision of 7/10 in the affected eyes, 5 developed an atrophy of the choroid and the retina, 7 showed retinal scars and a vision of 1/15 or less, 3 had only light perception and 3 became completely blind. (6 figures, 13 references) Ernst Schmerl.

Verrey, F. **A critical study into the causes of failure of retinal detachment operations.** Ophthalmologica **123**:271-276, April-May, 1952.

Between April, 1944, and May, 1951, 239 cases of retinal detachment were admitted to the Eye Clinic of the University of Zürich. Of these 202 patients were operated upon, 61.9 percent with complete and lasting reattachment; in 9.9 percent reattachment occurred but did not last, and in the remaining 28.2 percent the retina did not become reattached. A study

of the records of these failures revealed a number of factors which seemed related to the unfavorable outcome. Such factors were severe diffuse retinal degeneration in malignant myopia, giant tears, heavy vitreous strands especially after perforating injuries, aphakia, failure of the retina to settle down after bedrest with pinhole spectacles, early formation of radial folds in a yellowish-green, apparently thickened retina, centrally located rents or tears, and fixed folds in retinas of long standing. Advanced age and a poor state of health were also unfavorable factors. Despite the presence of one or several unfavorable factors a few patients did surprisingly well and were completely cured.

The operation, diathermy or catholysis, constitutes a severe trauma to the eye. In cases of extensive fundus degeneration and multiple tears the surgeon faces a real dilemma: an operation covering the entire diseased area entails serious dangers; an incomplete operation is almost always a failure. A "happy medium" does not exist. It is in these cases that the "newer methods" such as the scleral resection and air injections are indicated.

Peter C. Kronfeld.

12

OPTIC NERVE AND CHIASM

Lisch, K. **The role of the retina in retrobulbar neuritis.** Klin. Monatsbl. f. Augenh. **119**:55-61, 1951.

Clinical and pathologic evidence is convincingly presented to emphasize the fact that the primary lesion in retrobulbar neuritis lies in the peripheral pathways of the optic nerve and not in the retina. This paper is a reply to Poos (Klin. Monatsbl. f. Augenh. 116:159, 1950), who speculated on the retinogenic basis of retrobulbar neuritis caused by a vascular stasis in the choroid.

Frederick C. Blodi.

Nonnenmacher, Heinz. **Diseases of the optic nerve and the retina due to nutritional disturbances.** Klin. Monatsbl. f. Augenh. **119**:262-270, 1951.

In 13 persons who had developed malnutrition in prison camps, the ocular findings consisted of diminished central vision, central scotoma for white and colors, temporal pallor of the papilla and occasional peripheral contraction of the visual field. The condition resembled the so-called inanition amblyopia of Obel. (9 references)

Ernst Schmerl.

v. Wolffersdorff, H. **Pyrifer treatment of various diseases of the optic nerve from 1939-1950. Special consideration of macular affections in acute retrobulbar neuritis.** Klin. Monatsbl. f. Augenh. **119**: 271-279, 1951.

The author used the coli-vaccine pyrifer for the administration of a fever-treatment in 107 cases of optic neuritis. Focal infections of teeth, tonsils and sinuses were present in two thirds of all cases. Optic neuritis and atrophy due to lues, tumors or sclerosis of the blood vessels were excluded from this series. In eighty percent of all cases the visual function was recovered after treatment with 10 to 15 injections. (4 figures, 9 references)

Ernst Schmerl.

13

NEURO-OPHTHALMOLOGY

Alpers, B. J., Schlezinger, N. S., and Tassman, I. M. **Bilateral internal carotid aneurysm involving cavernous sinus.** A.M.A. Arch. Ophth. **46**:403-407, Oct., 1951.

The case of a woman, 45 years of age, with bilateral aneurysm of the portion of the internal carotid artery in the cavernous sinus is presented. She was known to have hypertension, and about 11 months before the onset of her present illness she

had weakness of the left arm for about two months. She had severe headache and complete ophthalmoplegia on the left side, followed in 30 days by headache and ophthalmoplegia on the right. Examination revealed bilateral exophthalmos with complete ophthalmoplegia. A loud bruit was heard over the right side of the face and head. Arteriographic studies revealed a carotid artery-cavernous sinus fistula on the right side and a saccular aneurysm on the left. No surgery was done. Later there was complete return of movement of the right eyeball except for residual paralysis of the external rectus muscle. The ophthalmoplegia on the left side remained unchanged. Bilateral aneurysms in the sinus are rare and present difficulty in diagnosis and treatment.

John C. Long.

Bonynge, T. W., and Von Hagen, K. O. **Severe optic neuritis in infectious mononucleosis; report of a case.** J.A.M.A. **148**: 933-934, Mach 15, 1952.

A well-marked optic neuritis complicated an acute infectious mononucleosis in a fourteen-year-old white girl. Visual reduction to a bare finger counting, dilated, non-responsive pupils and a considerable left hyperphoria were the other important eye signs. Although visual improvement had already started, marked, further, and more rapid improvement followed dimercaprol (BAL) injections. Early recognition of peripheral or central nervous complications of this disease, so that prompt dimercaprol or gamma globulin treatment may be started, will benefit the patient. F. M. Crage.

Cogan, David G. **Divergence paralysis and paralysis of the near reflex associated with a malnutrition syndrome (presumably Wernicke's polioencephalitis).** A.M.A. Arch. Ophth. **46**:436-437, Oct., 1951.

This is a case report of the condition described in the title. The patient was cachectic as the result of a major intestinal disorder. Practically complete recovery ensued. John C. Long.

Dufour, R., and Cuendet, J. F. **The assessment of visual damages.** Ophthalmologica **123**:249-255, April-May, 1952.

The visual acuity, the visual field, and the dark adaptation should be considered in calculating the percentage of visual loss due to optic nerve injuries. Any loss in the second eye is much more serious than in the first eye. A chart for easy calculation of the percentage of visual loss is presented. Peter C. Kronfeld.

Eareckson, V. O., and Miller, J. M. **Third-nerve palsy with sparing of pupil in diabetes mellitus.** A.M.A. Arch. Ophth. **47**:607-610, May, 1952.

Extraocular paralyses in diabetes are uncommon. Third-nerve palsy, with sparing of the pupil is rare, and is thought to be due to hemorrhage in the third-nerve nucleus. Because of the accompanying pain, such cases are frequently confused with aneurysm of the internal carotid. A case of extraocular muscle paralysis complicating long-standing diabetes is reported. R. W. Danielson.

Franceschetti, A., and Rickli, J. H. **Retrobulbar neuritis due to acute polyhalogen hydrocarbon intoxication.** Ophthalmologica **123**:255-260, April-May, 1952.

The case reported concerns a workman (apparently not a professional painter) whose job consisted of cleaning and painting the inside of three wine tanks. To prevent contact of the wine with the metal of the tank wall the latter was to be covered with a material called "Gemmex". The manufacturer's directions advised the use of a gas mask while handling the material. This precaution

however, was not observed. After several hours of exposure to the very unpleasant odor spread by the material in the poorly ventilated tanks, the workman fell asleep in the tank, was rescued half an hour later, regained consciousness promptly and was able to walk home the same evening. Several months later he began to notice visual impairment which proved to be due to a bilateral retrobulbar neuritis and atrophy with absolute central scotomas and general depression. Despite various forms of treatment the condition became stablized at a level of 20/200 in each eye. Polyhalogen hydrocarbons were identified in the solvent of "Gemmex". The case is interpreted as a retrobulbar neuritis due to acute polyhalogen hydrocarbon intoxication. Peter C. Kronfeld.

Gaines, Thomas R. **Postdiphtheritic cycloplegia.** A.M.A. Arch. Ophth. **45**:416-418, April, 1951.

Postdiphtheritic cycloplegia lasted two and one half months in one and seven weeks in the other of two schoolgirls. Bifocal lenses were worn temporarily with satisfaction until recovery was complete. John C. Long.

Huber, A. **The distinction between unilateral papilledema, pseudopapilledema and pseudoneuritis.** Ophthalmologica **123**:261-268, April-May, 1952.

The distinction between papilledema and pseudopapilledema is made by the neurosurgeon by determining the presence or absence of an elevation of the intraocular pressure. After all true papilledemas have been excluded, a heterogeneous group remains which consists of a good many cases of true pseudoneuritis (that is, congenital anomalies resembling neuritis) and a few cases of pseudopapilledema due to ill-defined, often obscure etiologies. Unilateral pseudopapilledema may be caused by deep-seated colloid bodies (Drusen) of the disc.

Peter C. Kronfeld.

Lijo Pavia, J., Arouh, J., and Lis, M. **The benign retinal pituitary syndrome.** Rev. oto-neuro-oftal. **25**:128-131, Dec., 1950.

This syndrome comprises fundus changes (retinal edema, pigment deposits), visual disturbances and osseous changes around the pituitary, visible in X-ray films. The author advises treatment with sex hormones.

Frederick C. Blodi.

Lijo Pavia, J., and Arouh, J. **Ophthalmoscopic aspects of optico-chiasmatic arachnoiditis.** Rev. oto-neuro-oftal. **25**: 99-104, Nov., 1950.

On the basis of four cases, the authors discuss the difficulties of diagnosis and differential diagnosis.

Frederick C. Blodi.

Smith, Vernon L. **Bilateral transverse optic neuritis.** A.M.A. Arch. Ophth. **46**: 363-366, Oct., 1951.

Severe transverse optic neuritis with bilateral blindness in men, 30 and 18 years of age is reported. One patient had transient urinary retention and alteration of reflexes, suggestive of mild myelitis, while the second patient had no positive neurological signs at any time. These cases may represent what has been called "abortive neuromyelitis optica". Cortisone was administered in each case and visual improvement was rapid. The part played by cortisone in limiting the progress of the disease is unknown, but this drug may be important in treatment.

John C. Long.

Trelles, J. O., and Roederbeck, S. D. **Total external ophthalmoplegia and cerebral signs caused by congenital syphilis.**

Rev. oto-neuro-oftal. **25**:41-43, March-May, 1950.

A 15-year-old girl had attacks of unilateral ophthalmoplegia with 7th nerve paresis and signs of increased intracranial pressure. Kahn and Wassermann tests were positive in the blood, but not in the cerebrospinal fluid. Frederick C. Blodi.

14

EYEBALL, ORBIT, SINUSES

Alvaro, M. D. **Extrusion of implant after 33 months in the orbit: study of the corrosion of its surface.** Tr. Am. Acad. Ophth. **56**:43-45, Jan.-Feb., 1952.

The author describes two cases in which Cutler type implants were extruded 33 and 30 months after operation. Photomicrographs of the surface of the plastic part of the implant show signs of corrosion, whereas the tantalum part does not. Extrusion of the implant is ascribed to some kind of reaction to the corroded plastic. (6 figures)

Theodore M. Shapira.

Bürki, E. **The clinical value of X-ray studies of the optic canal.** Ophthalmologica **123**:243-248, April-May, 1952.

In two cases of unilateral, slowly progressive visual loss the roentgenologic examination of the optic foramen according to the method of Rhese-Goodwin yielded valuable information (destruction of the lateral wall in the first and narrowing of the canal in the second case). Exploration by the neurosurgeon revealed large meningiomas in both cases.

Peter C. Kronfeld.

Cowan, Thomas W. **Orbital pseudotumors.** A.M.A. Arch. Ophth. **46**:390-402, Oct., 1951.

The term "pseudotumor" is used to designate any tumor of the orbit that is not a true neoplasm. The author discusses the etiology, pathology, symptomatology, diagnosis, classification and treatment of orbital tumors. A very interesting and involved case of pseudotumor of the orbit is described in a man of 24 years. Because of pain and dull frontal headache, a diagnosis of neuralgia was first made. Later, pain over the temporal artery and relief from the injection of procaine around the artery suggested temporal arteritis. Later multiple extraocular muscle paresis, ptosis, some proptosis and unilateral papilledema developed. A tumor mass was removed from the orbit by a transfrontal approach. This was diagnosed as chronic inflammation in tissue from the retrobulbar area. Later the globe became more or less fixed and displaced. A thrombosis of the central retinal vein occurred and a flat detachment of the retina developed. In view of the useless condition of the eye and the possibility of an orbital malignancy, an exenteration of the orbit was carried out. Pathological diagnosis of the excised tissue was chronic inflammatory pseudotumor. Plastic repair of the orbit produced a good cosmetic result.

John C. Long.

de San Martin, A. M., Arouh, J., and Cabrera, M. **Retrobulbar abscess in a premature infant.** Rev. oto-neuro-oftal. **25**: 36-38, March-May, 1950.

A premature infant (birthweight 1,600 grams) developed a unilateral orbital cellulitis at the age of one month. After treating the child with antibiotics for one day the symptoms became worse and the retrobulbar abscess had to be opened.

Frederick C. Blodi.

15

EYELIDS, LACRIMAL APPARATUS

Bischler, V. **A case of congenital pseudo-von Graefe phenomenon.** Ophthalmologica **123**:322-326, April-May, 1952.

A case of slight unilateral ptosis with a deep upper palpebral furrow in primary position, and some lid lag on looking down is interpreted as the result of a hereditary anatomic anomaly of the levator palpebrae. Peter C. Kronfeld.

Ertel, Rolff. **A supramid tube in stenosis of the lacrimal sac.** Klin. Monatsbl. f. Augenh. **119**:140-146, 1951.

The use of a plastic tube is advocated for cases of lacrimal stenosis. The tube is inserted as a connection between lacrimal sac and nasolacrimal duct.

Frederick C. Blodi.

Fox, Sidney A. **Relief of senile entropion.** A.M.A. Arch. Ophth. **46**:424-431, Oct., 1951.

Senile entropion is primarily due to the relative enophthalmos and general relaxation of tissues, rather than to localized spasm of the orbicularis muscle. A new operation is described and the results reported in its use in the repair of 14 lids in 10 patients. The entropion has been corrected and recurrences have not been observed. The surgical technique is as follows: The lateral half of the lid is split into a skin-orbicularis layer and a tarsal conjunctival layer. A triangle of tarsus is resected in the center of the lid with the apex up. A spindle of skin and orbicularis is resected just below the outer canthus. After suturing the defects thus caused, the entropion is overcome. (3 figures) John C. Long.

Galpine, J. F., and Walkowski, J. **A case of mumps with involvement of the lacrimal glands.** Brit. M.J. 1:1069-1070, May 17, 1952.

Sublingual and lacrimal gland swellings were noted at the same time, one day after the onset of malaise. The eyelid swelling became very severe on the third day but subsided with the salivary adenitis after the fourth day.

Robert A. Moses.

Robertson, E. Norris. **The treatment of dacryocystitis in infants.** South. M. J. **45**: 208-211, 1952.

The author advocates early probing of the congenitally obstructed tear duct. The majority of these patients were cured by one procedure; there was one failure in a child who had had the condition for eighteen months and had had no previous treatment. The probing is an office procedure. The infant is anesthetized and tetracain instilled into the conjunctival sac. A few passages may be opened by simply irrigating with saline, and this is attempted before the probe is inserted. Extreme gentleness, a small probe, and a knowledge of the anatomy of the passageway are requisites for success. A false passage was made twice; neither patients showed any ill effect. After perforating the membrane at the lower end of the nasolacrimal duct, a second probe is inserted through the nares under the inferior turbinate to check on the position of the operating probe. After removal of the probe, a lacrimal needle is inserted directly into the nasolacrimal duct for a distance of 0.5 cm. and irrigation with saline is performed. Aftercare includes the use of an antiseptic eye wash such as zephiranchloride, 1:5,000, for several days.

Under the circumstances, the author feels that the conservative treatment of such infants is undesirable. In his community, pediatricians realize the benefits of early probing, and refer the patients to the ophthalmologist at an early age. Reluctance to probe infantile passages may account for the popularity of conservative therapy. James W. Brennan.

16

TUMORS

Fuchs, Adalbert. **The clinical diagnosis of a choroidal hemangioma and its relation to the nervous system.** Rev. oto-neuro-oftal. **26**:99-108, Sept.-Oct., 1951.

Two types of choroidal hemangioma are recognized. One is a congenital malformation, leads early to blindness and may be accompanied with a facial hemangioma. The other type is acquired later in life, grows slowly and is not associated with a facial hemangioma. The author describes the clinical signs of a choroidal hemangioma and its relation to the Sturge-Weber syndrome.

There could be a causal connection between choroidal hemangioma, hydrophthalmos and von Recklinghausen's disease of the choroid. A relationship between hemangioma of the choroid and the nervous system was assumed by the author when he found ganglion cells in the choroid in six of 15 cases and in ciliary nerves behind the posterior pole of the globe in two. This finding is significant when compared with the rarity of ganglion cells in the choroid and in ciliary nerves in routine histologic sections. Frederick C. Blodi.

Pillat, A. **Incidence and course of metastatic carcinoma of the choroid in breast carcinoma of the male.** Wien. Klin. Wchnschr. **64**:222-224, March, 1952.

Of the 200 cases of metastatic carcinoma of the choroid, 65 percent occurred after carcinoma of the breast. Only three cases have been described in men. The author's own observation concerns a 46-year-old man who developed metastatic carcinoma of the choroid 40 months after removal of a primary breast cancer. Some vision was retained in this eye with X-ray treatment (6000 r). The patient died nine months later because of lung metastases. The second eye became affected five months after cancer was discovered in the first. Max Hirschfelder.

Spaeth, Edmund B. **Ocular tumors.** A.M.A. Arch. Ophth. **46**:421-423, Oct., 1951.

A statistical analysis of 170 consecutive cases of ocular tumors is presented in six tables. The only certain conclusion one can draw from this analysis is that patients with sarcoma die early in life, and early in the course of the disease, and that adenocarcinoma is the most malignant of all the epithelial-cell tumors of the eye. John C. Long.

17

INJURIES

Lijo Pavia, J. **The effect of irradiation on the eye.** Rev. oto-neuro-oftal. **26**:25-36, March-April, 1951.

In this survey the author discusses the effects of electro-magnetic energy on the eyeball. Ultraviolet and infrared radiation are treated in detail. Their importance as an occupational hazard and their prophylaxis are discussed.

Frederick Blodi.

Löhlein, W. **Operation for intraocular foreign body in the only eye.** Klin. Monatsbl. f. Augenh. **119**:113-121, 1951. 1951.

On the basis of his experience the author advises conservative treatment in some cases of intraocular foreign bodies. This is especially true if the foreign body is glass, which remains inert. It is also true for alloys of copper which do not necessarily lead to a sterile abscess as the alloys used in this World War contained little copper. It can even be tried with iron, if the particle is small, well encapsulated and could only be extracted with great danger.

Frederick C. Blodi.

18

SYSTEMIC DISEASE AND PARASITES

Appelmans, M. **Visual disturbances in trypanosomiasis, leprosy, and onchocerciasis in the lower Congo.** Arch. d'opht. **11**:729-738, 1952.

Appelmans concludes from survey figures that blindness is more common in the Congo than in Belgium and estimates its occurrence at between one and two per thousand. Protozoan and microbian causes of blindness are the most common; ophthalmia neonatorum is rare, variola is disappearing, and trachoma is unknown. Syphilis, tuberculosis, the meningitides, and the encephalitides are important causes of blindness, and trypanosomiasis, leprosy, and onchocerciasis are still of major importance.

The author discusses the diagnosis of trypanosomiasis by lymphnode and lumbar puncture, centrifugation of blood, and cytological changes in the blood. In 1947 there were 21,969 cases discovered among 3,000,000 natives but the incidence seems to be diminishing. Visual complications occur in 20 to 25 percent and optic atrophy from arsenical treatment is common. In 1947, 61,000 lepers were discovered. The visual complications of leprosy are described in detail and are reported as milder among the native Bantous than among white individuals. Onchocerciasis was found to be more common than trypanosomiasis and leprosy. Diagnosis is based on the finding of nodules or on the microscopic identification of the parasites in the intercellular fluid after scarification of the skin; rarely have the organisms been found in the anterior chamber on biomicroscopic examination. The author describes the wide variety of ocular lesions produced, which vary from optic neuritis to superficial keratitis. Treatment by extirpation of the nodules and administration of hetrazan or notezine has been partially successful. When blindness ensues, it is chiefly because of lesions of the posterior segment. P. Thygeson.

Bartelheimer, Heinrich. **Modern trends in endocrinology.** Klin. Monatsble. f. Augenh. **119**:225-240, 1951.

In this survey lecture given at a meeting of ophthalmologists the author gives an excellent exposition of the more recent achievements of the increasingly important science of endocrinology. Blindness due to a diabetic retinopathy seems to be the most frequent and most dangerous condition of ophthalmologic interest produced by endocrine disturbances. However, hyperinsulinization has also to be considered where retinal hemorrhages occur. This view is in agreement with the experiences of some ophthalmologists who reported a bloodsugar level of 140 to 150 mg. percent as most desirable.

Ernst Schmerl.

Dunphy, E. B. **Some external ocular signs associated with constitutional disease.** South. M. J. **45**:202-208, 1952.

In this article, the author has selected several systemic diseases, and reviews the associated external eye findings.

One of the most important diseases for the ophthalmologist to consider is hyperthyroidism. Lid findings are frequent, and often disappear following thyroidectomy. This may give a false sense of security, as there may be an actual increase in the exophthalmos after surgery. The increase is not apparent however, because of the reduction of lid retraction. This is generally observed in the thyrotoxic type of exophthalmos. Fullness of the lids, chemosis, enlarged venous sinuses over the lateral rectus muscles, and a definite resistance upon attempting to push the globe back into the orbit are characteristic of thyrotropic exophthalmos and may be followed by corneal ulceration

and diplopia. In contrast, in the thyrotoxic type the globe offers little resistance and can be readily pushed into the orbit, and lid swelling is not a prominent feature.

Ptosis is one of the first signs to appear in myasthenia gravis. Disturbances of extraocular muscles are found in both myasthenia and multiple sclerosis. Any nodular enlargement of the lid or eyeball, particularly in a negro, should suggest sarcoidosis. Band keratopathy occurring in otherwise normal eyes may indicate hypercalcemia from hyperparathyroidism or excessive intake of vitamin D. It consists of superficial paralimbal opacification extending axially but avoiding the pupil. There may be an associated nephrocalcinosis. The relationship between riboflavin deficiency and corneal vascularity is over-emphasized although the author has seen a patient so affected. Other diseases discussed are syphilis, tuberculosis, trichinosis, lipoid disturbances and exanthemata.

James W. Brennan.

Linnen, H. J. **External ophthalmomyiasis.** Klin. Monatsbl. f. Augenh. **119**:19-23, 1951.

A case of external myiasis was treated with the new Mintacol, which originally was synthetized as an insecticide. These drops soon paralyzed and loosened the larvae.

Frederick C. Blodi.

Rodger, F. C. **Nutritional amblyopia.** A.M.A. Arch. Ophth. **47**:570-583, May, 1952.

This is a statistical study of the course and progress in 238 cases. All the patients were former Far-Eastern prisoners of war and the visual histories extended over a period of more than ten years. They were seen after release in the subacute and chronic stages. The relative sick discharge rate of the repatriated prisoners of war was 6.8 percent; this does not include those who recovered or died. The visual acuity is markedly reduced; practically all the patients had changes in the field and 64 percent of these were central scotomata. About 10 percent were pericentral and 1.6 percent centrocecal. The appearance of bitemporal pallor was common, probably due to changes in the capillaries on the disc. The fundus was anemic, which tends to obscure the appearance of the temporal pallor.

Even after return to a normal diet, about a third of 211 patients followed, deteriorated, a third improved, and a third remained unchanged. The pericentral scotomata remained comparatively unchanged, but the slight temporal pallor seen in the subacute stage advanced to a well-demarcated optic nerve atrophy. With a high carbohydrate diet, low in all the respiratory enzyme vitamins, the ganglion cells in the central areas of the retina underwent degeneration. The author believes the damage is done by an endogenous toxin. (12 tables, 1 figure, 24 references) R. W. Danielson.

Sherrill, J. W. **Diabetic retinitis: the relationship between retinal degenerative changes and the degree of diabetic control.** Bull. Scripps Metabolic Clin. **2**:1-17, Oct., 1951.

Failure in the treatment of diabetes is due mainly to the fact that the disease is painless and the patient complains only when complications appear. Two groups of diabetics were studied. In one the disease had appeared after 18 years of age and was present for 20 to 38 years; in the other the disease had appeared before the age of 17 and had been present for 20 to 30 years. The progress of the disease in these cases is illustrated by general, laboratory, and ocular findings.

By proper control of the diabetes the

number of the capillary aneurysms was reduced and the vision of many of the patients was improved. To avoid failure in its control, diabetes should be considered a disease of total metabolism rather than one of defective carbohydrate metabolism only. It is wrong to adjust diets to normal caloric requirements. Reduction in weight of the obese is important and those who are underweight should not be allowed to become fat. Short-cut methods and inadequate administration of insulin are harmful procedures. F. M. Crage.

Thomas, C., Cordier, J., and Algan, B. **Oculoglandular tularemia.** Arch. d'opht. 12:19-41, 1952.

During an epidemic of tularemia in the Lorraine region of France during the winter of 1949-50, the authors were able to observe 75 of the 112 known cases. Of these 75 cases, four were of the oculoglandular type. The history and clinical course of the disease in these four patients are given in detail. Rabbits were the source of the infection, and the patients were rural workers. All had fever and preauricular adenopathy after an incubation period varying from five to ten days. Onset was acute with violent conjunctival inflammation. The disease was unilateral; the conjunctival lesions were nodular and ulcerative and thin pseudomembranes were observed. Secretion was scanty. Healing occurred spontaneously in from four to five weeks. There was preauricular and submaxillary adenopathy, painful mastication and suppuration.

The authors discuss the problem of clinical diagnosis, particularly in the occasional mild, atypical case, and stress the importance of a history of rabbit-handling. They describe the technique of isolation of Pasteurella tularensis and of the various serological tests and the allergic skin test. The antibiotics streptomycin and aureomycin are the medications of choice. P. Thygeson.

Venturi, G. **The ocular-muco-cutaneous syndromes, particularly a very severe case of Stevens-Johnson disease.** Boll. d'ocul. 31:21-40, Jan., 1952.

A 16-year-old girl had been seen for six years because of an intractable condition of the skin, mucous membranes, and eyes, which finally led to bilateral blindness. From clinical and pathologic studies the author concluded that Stevens-Johnson syndrome should not be identified with either erythema exudativum polymorphum or erythema exudativum multiforme. Diathermy coagulation of the lacrimal canaliculi gave relief from the very distressing symptoms of xerosis. Tests for psittacosis and lymphogranuloma inguinale were performed to rule out a possible virus etiology (six years after the onset of the disease) and proved to be negative. (6 figures, 140 references) K. W. Ascher.

19

CONGENITAL DEFORMITIES, HEREDITY

Falls, H. F., and Neel, J. V. **Genetics of retinoblastoma.** A.M.A. Arch. Ophth. 46:367-389, Oct., 1951.

An attempt was made to find all cases of retinoblastoma occurring in the state of Michigan between 1938 and 1947. Fifty-three cases occurred in 52 families. The incidence is approximately 1 for each 20,288 live births. Because the disease is fatal, the gene responsible for the condition is under strong negative selection pressure. That the condition persists at all is ascribed to the appearance in each generation, as the result of mutation, of new genes determing the development of the disease. The rate of mutation to this gene is calculated to be 2.3×10^{-5} per generation. It is estimated that the five-year survival rate of patients is near 70 percent. John C. Long.

Huber, O. **The practice of ophthal-**

mology in the canton Glarus. Ophthalmologica **123**:189-201, April-May, 1952.

Because of its isolation, partly geographic and partly traditional, the population of the canton of Glarus is most suited for the study of the inheritance of ocular diseases. The pedigree of a large glaucoma family is reported.

Peter C. Kronfeld.

Müller, F. **A family tree of Leber's disease.** Klin. Monatsbl. f. Augenh. **119**:62-76, 1951.

In a family tree of 103 persons in five generations, 22 persons (13 men and 9 women) were affected. The heredity was autosomal dominant with a changing penetration. Frederick C. Blodi.

Tiberi, Gian Franco. **A case of double lower lacrimal punctum.** Ann. di ottal. e clin. ocul. **78**:9-16, Jan., 1952.

Tiberi reports a case of a supernumerary lower left lacrimal punctum which communicated with a normal canaliculus. Embryogenetic and histologic studies support the hypothesis of the essential identity between so-called congenital lacrimal fistulas and certain congenital malformations of the tear passages. (15 references)

Harry K. Messenger.

20

HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Hurley, John. **The reading development laboratory.** U. S. Armed Forces M. J. **2**: 1915-1919, Dec., 1951.

This laboratory, a part of a medical field service school, was established to improve the reading habits of medical officers to the point where their reading skill becomes commensurate with the requirements of their positions.

With promotion goes a heavier reading load. This calls for increased reading speed with an associated increase in comprehension and retention. The course includes 31 hours of work over a period of 15 weeks. The time is spent with the tachistoscope, the reading rate controller and in group classroom study. A table shows the increase in reading speed and comprehension which members of the eleven classes who have completed the courses have developed. F. M. Crage.

Johnson, Victor. **Problems in graduate medical education.** Tr. Am. Acad. Ophth. **56**:7-15, Jan.-Feb., 1952.

To meet the needs of physician veterans of World War II, facilities for medical graduate training expanded to meet their needs, and have continued to expand, so that there are now about four times as many residencies as there were ten years ago. One fourth of these residencies are vacant. The outlook is for further increases in unoccupied residencies since the number of approved residencies exceeds, by five times, the number necessary to replace the specialists who die. (7 tables, 20 references)

Theodore M. Shapira.

Neuenschwander, M. **Homeopathy in ophthalmology.** Ophthalmologica **123**:316-320, April-May, 1952.

The author is a practicing ophthalmologist in Zürich who uses homeopathic methods where the methods of orthodox, that is allopathic, medicine have failed. He reviews the general principle and some of the specific ophthalmological methods of homeopathy. To quote an example, asthenopia not relieved by careful refractive measures and correction of hetereophorias is treated orally with a very weak solution of phosphoric acid, five drops three times daily. Peter C. Kronfeld.

NEWS ITEMS

Edited by DONALD J. LYLE, M.D.
601 Union Trust Building, Cincinnati 2

News items should reach the editor by the 12th of the month but, to receive adequate publicity, notices of postgraduate courses, meetings, and so forth should be received at least three months before the date of occurrence.

DEATHS

Dr. Samuel Davidson Evans, Pittsburgh, Pennsylvania, died June 1, 1952, aged 51 years.

Dr. William Davis Gill, San Antonio, Texas, died May 24, 1952, aged 56 years.

Dr. William Aloysius Kennedy, St. Paul, Minnesota, died April 30, 1952, aged 58 years.

Lieut. Col. Jacques Duquet, stationed at Friedrichshaven, Germany, died June 24, 1952, aged 44 years.

ANNOUNCEMENTS

LOUIS BOTHMAN FELLOWSHIP

Establishment of the Louis Bothman Fellowship in ocular pathology at the University of Illinois College of Medicine has been announced by Dean Stanley W. Olson.

The university has received a grant in the amount of $5,000 from the Louis Bothman Memorial Fund, Inc., of Phi Delta Epsilon fraternity in support of the fellowship in ophthalmology.

The late Dr. Bothman held the rank of clinical professor of ophthalmology at the University of Illinois. He was instrumental in developing a laboratory in ocular pathology at the University's Research and Educational Hospitals.

CANCER PROGRAM FOR FISCAL YEAR 1953

The U. S. Atomic Energy Commission, in the fiscal year 1953, is changing its previous policy of distributing radioisotopes for cancer research and therapy free of production costs. Instead, it will make a charge of 20 percent of production costs. This change in policy affects only the price and not the availability of isotopes; production will continue to meet all demands.

The following table shows the prices per millicurie which will be charged, beginning July 1, 1952, for the four most important radioistopes used in cancer research or therapy, compared with prices for noncancer use:

	Cancer use	*Noncancer use*
Radioiodine 131	$0.15	$ 0.75
Radiophosphorus 32	0.22	1.10
Radiogold 198	0.05	0.24
Radiocarbon 14	7.20	36.00

Under the new policy, radioisotopes will be distributed by the Atomic Energy Commission and Atomic Energy Commission distributors from July 1, 1952, through June 30, 1953, at a reduction of 80 percent from the AEC catalog price, within the following limitations:

Cancer research. All radioisotopes will be available at the reduced price for this purpose. Cancer research is interpreted to mean the following: (1) Investigations of the basic aspects of normal and abnormal cellular growth; (2) development and evaluation of therapeutic and diagnostic procedures for cancer and allied diseases.

Cancer diagnosis and therapy. Radiophosphorus 32, Radioiodine 131 and Radiogold 198 will be distributed at the reduced price for use in routine or nonexperimental diagnosis and treatment of cancer and allied diseases. (Radioisotopes for treatment of nonmalignant diseases—such as hyperthyroidism and cardiac decompensation—are *not* included in this program. Separate application forms should be submitted to obtain materials for noncancer uses). Radioactive material for fixed sources such as Co 60 and Cs 137 are not included under this arrangement.

YALE CONFERENCES IN OPHTHALMOLOGY

The Yale University School of Medicine announces the program for its 1952-53 postgraduate course in ophthalmology:

1952

October 10: Case presentation—"Exophthalmos," Dr. R. M. Fasanella, New Haven, Connecticut.

October 31: "Rheumatism and the eye," Dr. Joseph Igersheimer, Boston.

November 14: Case presentation—"Diabetes and eye photography," Dr. Ernest Rosenthal, Hartford, Connecticut.

December 5: "Regeneration of surgical wounds after intraocular operations," Brittain Ford Payne, New York.

December 12: Case presentation—"The management of complications in cataract surgery," Dr. Arthur M. Yudkin, New Haven.

1953

January 9: Case presentations, Dr. Francis P. Guida, New Haven.

January 30: Round table discussion of "Surgical problems and their management," Dr. Edmund B. Spaeth, Philadelphia, chairman.

February 13: Case presentations, Dr. Clement C. Clarke, New Haven.

February 27: "Surgery of the lacrimal mechanism," Dr. Byron Smith, New York.

March 13: Case presentations, Dr. Frederick A. Wies, New Haven.

March 27: "Retinal detachment," Dr. Charles A. Perera, New York.

April 10: Case presentation—"Lacrimal gland tumors with discussion of the pathology." Dr. David Freeman, New Hoven.

April 24: "Suspensory ligament of Zinn and the zonular chamber," Dr. Henry Minsky, New York.

May 8: Case presentation—"The general surgeon in eye lesions," Dr. William H. Ryder, New Haven.

May 22: "Differential diagnosis of hemianopia," Dr. Alfred Kestenbaum, New York.

Societies

League against trachoma

Participating in the program at the 1952 meeting of the League against Trachoma, held in Paris in June were: Prof. G. Favaloro, Catania, Italy; Prof. Yukihiko Mitsui, Kumamoto, Japan; Dr. J. Taborisky, Haifa, Israel; Dr. Emile Olah, Gyula, Hungary; Prof. G. B. Bietti and Prof. F. Ferraris de Gaspare, Parma, Italy; Prof. Jean Charamis, Athens, Greece; Prof. M. Sohby, Pasha, Cairo, Egypt; Dr. D. Rapisarda, Catania, Italy; Dr. G. Guerguerian, Beyrouth, Lebanese Republic; Dr. Jean Sedan, Marseille, France; Dr. J. Carli, Haiphong, Indo-China; Dr. N. Monciino, Catania, Italy; Dr. G. Farnarier and Dr. G. Sobrepere, Marseille, France; Dr. A. Dauthuile, Lille, France; Dr. Svetozar Postic, Novi-Sad, Yugoslavia; Dr. H. Sakon, Casablanca, Morocco; Dr. V. Tabone, St. Julians, Malta; Dr. L. Poleff, Rabat, Morocco; Dr. G. Scuderi and Dr. S. Bellomio, Catania, Italy; Dr. F. Malnou, Tozeur, Tunisia; Dr. S. Etzine, Johannesburg, South Africa.

Instituto Penido Burnier

Officers of the Medical Association of the Instituto Penido Burnier, Campinas, São Paulo, Brazil, for the year 1952-53 are: President, Dr. F. J. Monteiro Sales; 1st secretary, Dr. Alberto Galo; 2nd secretary, Dr. Alberto Af. Ferreira; treasurer, Dr. L. de Souza Queiroz; directors of the *Archives*, Dr. Penido Burnier, Dr. Gabriel Porto, and Dr. Franco do Amaral.

Memphis meeting

The annual convention of the Memphis Eye, Ear, Nose and Throat Society will be held on February 7, 8, and 9, 1953. The guest speakers will be: Dr. Louis H. Clerf, Philadelphia; Dr. Albert Furstenberg, Ann Arbor, Michigan; Dr. Francis L. Lederer, Chicago; Dr. Albert D. Ruedemann, Detroit; Dr. C. Wilbur Rucker, Rochester, Minnesota; Dr. Willis S. Knighton, New York. For further information please write: Dr. Roland H. Myers, secretary, Exchange Building, Memphis 3, Tennessee.

Washington officers

The new officers for the Section on Ophthalmology of the Medical Society of the District of Columbia are:

President, Dr. Joseph Dessoff; vice-president (program), Dr. J. Spencer Dryden; secretary-treasurer, Dr. Robert L. Norment; executive committee, Dr. Everett S. Caldemeyer and Dr. William D. Foote.

Meetings will be held on November 4, 1952, and January 6 and May 5, 1953. These dates fall on the first Tuesday of the month.

Central Illinois Program

At the 19th convention of the Central Illinois Society of Ophthalmology and Otolaryngology held recently in Springfield, the following program was presented:

Dr. Edward C. Albers, Champaign, "Hemangioma of the orbit: A report of two cases"; Dr. Clarence A. Fleischli, Springfield, "The type of incision and sutures in cataract surgery"; Dr. Paul A. Chandler, Boston, Massachusetts, "Narrow-angle glaucoma," "Complication of cataract surgery," and "Choice of operation in convergent squint"; Dr. Thomas C. Galloway, Evanston, "Respiratory obstructions and their secondary effects," "Anoxia otolaryngology," and "Bulbar poliomyelitis and other secretional obstructions"; Dr. G. A. Rawlins, Alton, "Thornwaldt's disease."

Officers of the Central Illinois Society are: Dr. G. C. Otrich, Belleville, president; Dr. Philip R. McGrath, Peoria, president-elect; Dr. George H. Woodruff, Joliet, vice-president; Dr. Alfred G. Schultz, Jacksonville, secretary-treasurer.

Wisconsin-Michigan meeting

At the September meeting of the Central Wisconsin-Upper Michigan Society of Ophthalmology and Otolaryngology held at Bailey's Harbor, Door County, Wisconsin, the guest speakers were Dr. Noah D. Fabricant, Chicago, who spoke on: "Current trends in medication of the ear, nose, and throat" and "Amusing quotations for doctors and patients," and Dr. William F. Hughes, Jr., Chicago, who presented papers on "Repair of lid injuries, including entropion and ectropion," and "Present day practice regarding orbital implants after enucleation." Dr. Hughes also showed a movie on "The technique of implantation of buried polyvinyl implants."

Schoenberg Lecture

Dr. Charles L. Schepens of the Howe Eye Laboratory, Boston, will deliver the sixth annual Mark J. Schoenberg Lecture at the New York Academy of Medicine, New York, on Monday, December 1. The title of his address will be: "The prognosis and treatment of retinal detachment."

This lectureship, sponsored jointly by the National Society for the Prevention of Blindness and the New York Society for Clinical Ophthalmology, was established as a memorial to the late Dr. Schoenberg.

Personals

Dr. Jonas S. Friedenwald, Baltimore, delivered the Donders Lecture at a meeting of the Netherlands Ophthalmological Society held in Holland on September 20th and 21st. The subject of Dr. Friedenwald's address was "Studies on diabetic retinopathy."

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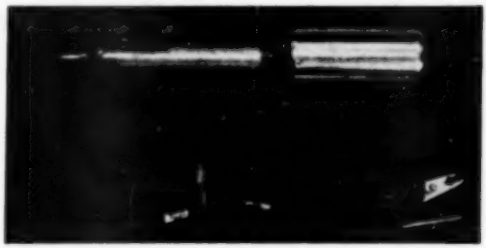

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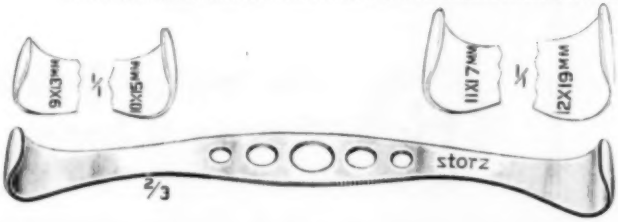

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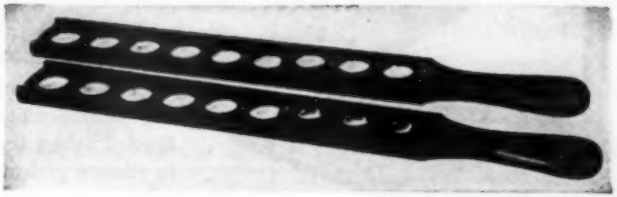

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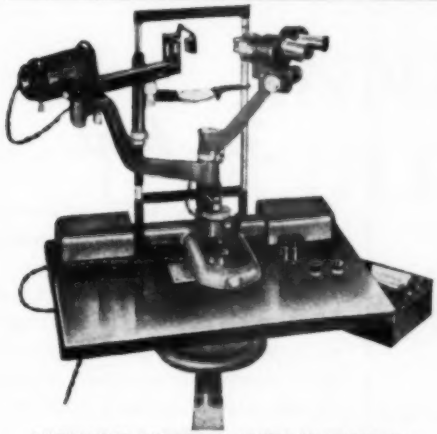

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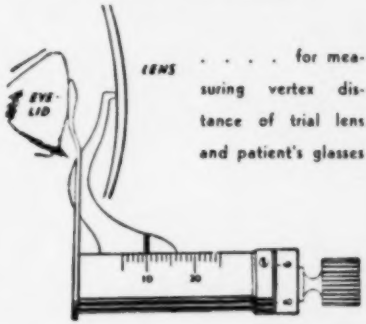

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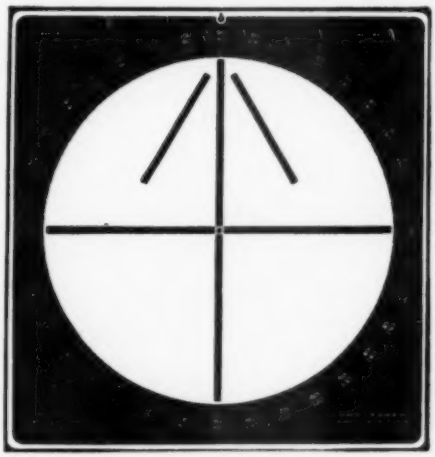

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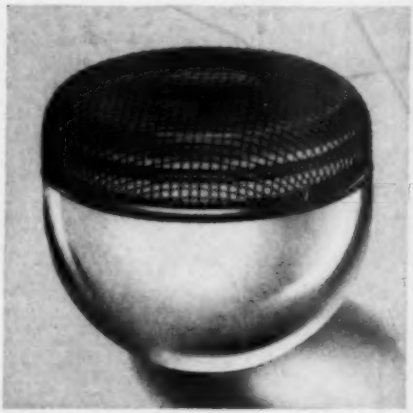

Fig. 1 *The Monoplex Buried Implant, in methyl-methacrylate or Kel-F, can be furnished with flat or gently rounded anterior surface.*

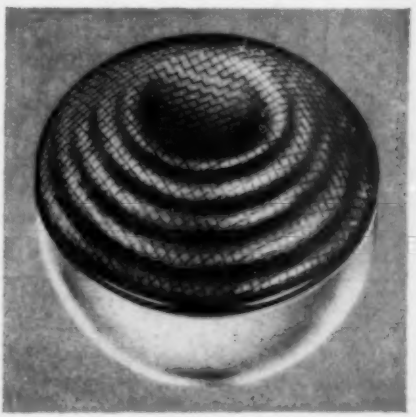

Fig. 2 *Fine tantalum mesh covers anterior surface. Grooves in surface provide passage for needles when suturing recti muscles to mesh.*